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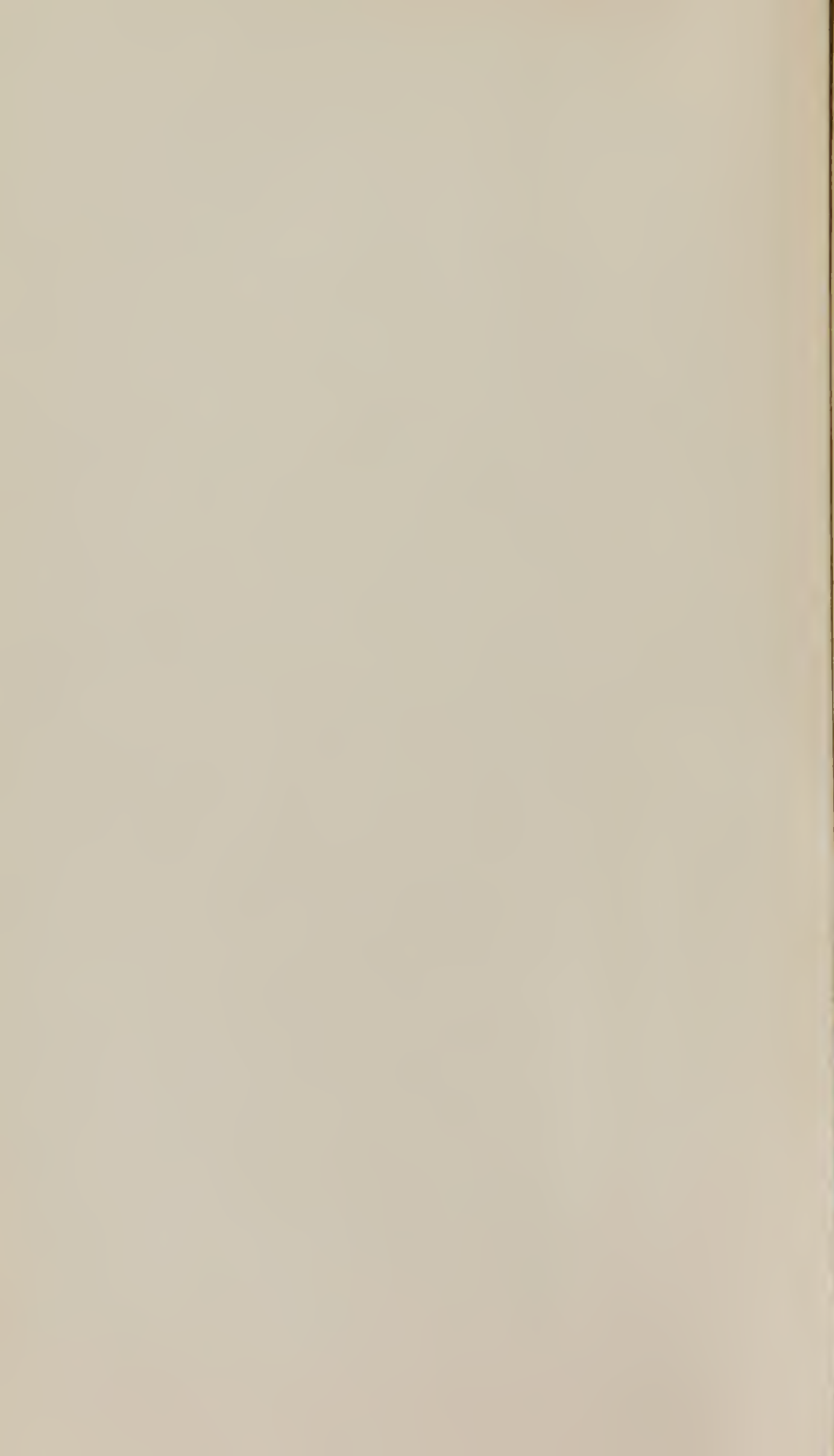
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AN ABSTRACT

—OF A—

COURSE OF LECTURES

—ON THE—

PRACTICE OF MEDICINE

—BY—

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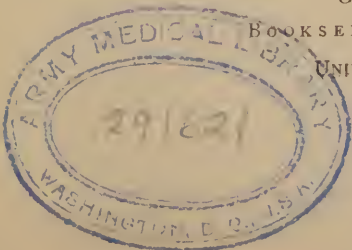
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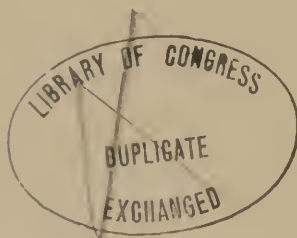


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PREFACE.

This Abstract is not intended to take the place of a regular text-book on the Practice of Medicine, but merely to assist the student to a better understanding of the *principles* of the subject of which it treats. I have therefore endeavored to make it *explanatory* as far as was possible in such a limited space.

CHAPTER I.

GENERAL PATHOLOGY.

Definition.—*General Pathology* treats of the morbid conditions which are common to many different diseases.

It is used in contradistinction to *Special Pathology* which treats of individual diseases.

DISTURBANCES OF THE CIRCULATION.

ANÆMIA.

Varieties.—1. *General*, meaning literally a deficiency of the *whole amount* of the blood, but usually used to denote a deficiency of the *red corpuscles*.

2. *Local*, meaning a partial or complete arrest of the blood supply to a limited part of the body.

General anæmia will be considered hereafter.

Causes of Local Anæmia.—1. A diminution in the lumen of the artery supplying a part with blood; such diminution may be due to one of the following conditions:

(1) disease of the wall of the blood vessel, such as atheroma, calcification, or syphilis;

(2) pressure on the vessel from without by tumors, inflammatory exudations, &c.;

(3) plugging of the vessel (thrombosis and embolism);

(4) Contraction of the walls of the vessels, due to cold, heat, ergot and other medicines, counter-irritation, &c. This contraction is usually brought about through the mediation of the nervous system.

2. Dilatation of the vessels in one part of the body causing increased flow of blood into that part and consequently a *diminution* in the amount of blood elsewhere. This is called *collateral anæmia* and is of very great importance from a therapeutic standpoint.

Results.—The results depend on the part involved and on the nature of the obstruction, but the general results are:

1. *Pallor*, from deprivation or diminution of red blood.

2. *Coldness*, because little or no oxygen is carried to the tissues and less heat is formed, and also because the warm blood itself is present in less amount than usual, or is cut off altogether.

3. *Defective nutrition*, because the nutritive materials are carried in the arterial blood and a diminution in the quantity of blood causes defective nutrition and often atrophy.

4. *Collateral circulation*, if the artery above and below the obstruction gives off anastomatic branches.

5. *Infarction*, if the vessel is a terminal one as in the brain, kidneys, lungs and spleen.

6. *Necrosis or gangrene*, when the blood supply is more or less completely cut off and certain germs gain access to the part thus deprived of blood.

7. Simple *softening*, when the blood supply is more or less completely cut off and the germs do *not* gain access to the anæmic part.

HYPERÆMIA.

Definition.—An abnormally large amount of blood in a part or organ

Varieties.—1. Active or arterial.

2. Passive, venous, or mechanical.

ACTIVE HYPERÆMIA.

Condition of the Vessels and Circulation.—The *arteries* are dilated and the blood flows more rapidly.

Causes of Active Hyperæmia.—A. *Diminished resistance* of the walls of the blood vessels caused by

1. Anything which weakens or paralyzes the muscular coat of the smaller arteries, such as

(1) sudden removal of pressure, as in ascites ;

(2) warmth ;

(3) paralysis of vaso-constrictor nerves from pressure, injuries, certain drugs, as amyl and alcohol, reflex agencies and also disturbances of nerve centres ;

(4) exhaustion from fatigue or over stimulation.

2. Irritation of vaso-dilator nerves as in certain cases of neuralgia.

B. Unusually great pressure in the vessels from increased force of the heart, which may be due to

(1) exertion ;

(2) certain stimulants, such as alcohol ;

(3) certain diseased conditions of the heart (hypertrophy).

C. Contraction of the blood vessels in one part of the body causing a dilation elsewhere. Hyperæmia caused in this way is called "*collateral hyperæmia*."

Results of Active Hyperæmia.—1. *Increased heat and redness* from increased amount of warm blood and increased oxidation in the part.

2. *Increased functional activity*, from increased supply of nutritive materials and also from elevated temperature.
3. *Possibly increase in connective tissue*, if of long duration.

PASSIVE HYPERÆMIA.

Condition of the Vessels and Circulation.—There is an excessive quantity of blood in the veins and capillaries and the flow is retarded.

Causes of Passive Hyperæmia.—1. Those which diminish the *vis a tergo*, as

- (1) weakness of the heart from any cause;
- (2) any impediment to the circulation in the arteries, as loss of elasticity or diminished calibre;
- (3) pressure on the capillaries by exudations or by effusions;
- (4) anything which interferes with the action of the valves in the veins as varicose veins, and certain affections of the thoracic or respiratory organs.

2. Those which *directly* interfere with the flow of venous blood through the veins, such as

- (1) pressure on the veins by the enlarged uterus, collections of fluid, new formation of connective tissue (as in cirrhosis of the liver), &c.
- (2) the plugging of a vein by a thrombus.
- (3) certain obstructive or regurgitative lesions of the valves of the heart.
- (4) certain pulmonary diseases (as emphysema) causing destruction of blood vessels in the lungs and consequent over-distension and dilatation of the right cavities of the heart and then of the veins.

Results of Passive Hyperæmia.—1. *Diminished velocity of blood current*, due to diminished pressure behind or to obstruction in front.

2. *Transudation of serum* from the blood vessels into the tissues, the transudation being due partly to increased intra-vascular pressure, but chiefly to a change in the vascular walls from defective nutrition, by which they are rendered more permeable.

3. *Escape of red corpuscles* from the vessels, due to the same causes as the transudation of serum, and *pigmentation* from the breaking up of these corpuscles and the deposit of their pigment in the tissues.

4. *Hæmorrhage*, from defective nutrition of the walls of the vessels which in connection with the over-distension causes them to rupture. Hæmorrhage from the stomach in cirrhosis of the liver is to be explained in this way.

5. *Fibroid induration*, due to the escape of white blood corpuscles from the vessels and the formation of connective tissue.

6. *Thrombosis*, the coagulation being due to a change in the vascular wall from defective nutrition.

7. *Necrosis, or Gangrene*, from the action of the germs of putrefaction on a part whose vitality is very much below the normal standard, in consequence of a defective supply of *arterial* or *nutritive* blood.

8. *Atrophy*, from an insufficient supply of arterial blood.

9. *Collateral circulation*, from enlargement of anastomosing veins above and below the seat of obstruction.

10. *Diminished heat* in the part from stagnation of blood and defective oxidation.

11. *Livid color*, from accumulation of venous blood.

12. *Diminished functional* activity from want of arterial blood.

DROPSY.

Definition.—The accumulation of fluid in the various tissues and cavities of the body; such fluid having passed out of the vessels by simple transudation and not in consequence of inflammation.

Varieties.—1. *Œdema*, or dropsy of the connective tissue.

2. *Anasarca*, a generalized œdema.

3. *Ascites*, dropsy of the peritoneal cavity.

4. *Hydrothorax*, dropsy of the thoracic cavity, &c., &c.

Causes of Dropsy.—1. Excessive flow of fluid from the vessels due to

(1) mechanical hyperæmia;

(2) a feeble and relaxed state of the vessels and tissues rendering the former more permeable;

(3) unhealthy condition of the blood which acts by rendering the blood more dilute and also by causing defective nutrition of the vascular walls;

(4) defective nervous influence (as in nettle rash);

2. Defective absorption by the veins and lymphatics in consequence usually of some obstruction to the flow through them.

Physical Character and Chemical Constituents of dropsical effusions—1. *Color*, usually pale straw color, rarely red from the presence of red blood corpuscles.

2. *Consistence*, fluid, and remains a fluid, differing in this respect from some forms of *inflammatory* exudate.

3. *Chemical constituents*; the fluid contains albumen and salts but the fibrin factors are present in small amount, if at all.

Clinical Forms of Dropsy.—1. *Cardiac*, from weakness of

heart or valvular lesions leading to passive hyperæmia. Dropsy appears first, as a rule, in the feet and ankles.

2. *Obstructive*, (as ascites from cirrhosis of the liver) due to mechanical hyperæmia and transudation from the veins behind the obstruction.

3. *Renal*, from change in the quality of the blood which becomes more watery and thus transudes more easily, and also causes defective nutrition of the vascular walls. Occurs in loose tissues, as eyelids.

4. *Cachectic*, from weakness of the heart and impaired nutrition of the wall of the blood vessels.

5. *Nervous*, of which no satisfactory explanation can be given.

THROMBOSIS.

Definition.—The coagulation of the blood at some part of the blood vascular system, the clot remaining where it is formed.

Seats.—A thrombus may form in the heart, arteries, capillaries or veins; it is most usually found in the veins.

Causes of Thrombi.—1. *Disease or loss of the endothelial lining of the vessels*, from injuries whether mechanical or chemical, inflammation (as in endo-carditis), defective nutrition of the vascular walls.

2. *Feeble circulation*, which not only causes defective nutrition of the walls of the vessels, but makes the blood more liable to coagulate from stagnation.

3. *Certain abnormal states of the blood itself*, which cause defective nutrition of the walls or in which there may be an increase of the fibrin factors.

4. The presence of foreign bodies in the vessel such as pieces of calcareous matter, cancer, &c.

Varieties of Thrombi.—1. *Red*, usually caused by the coagulation of the whole mass of blood at once.

2. *White*, usually formed gradually by the deposit of *fibrin* and *blood plaques* in successive layers.

Differences Between Thrombi and Post-Mortem Clots.

<i>Thrombi.</i>	<i>Post-Mortem Clots.</i>
<i>Color</i> , dark throughout or white and laminated.	<i>Color</i> , dark, with buff layer of greater or less thickness on the surface.
<i>Adherent</i> to the wall of the heart or vessels.	<i>Not</i> adherent to walls of the heart or vessels.

Later Changes in Thrombi.—1. *Decolorization*, from breaking up of the red corpuscles and absorption of their coloring matter,

2. *Resolution* or *absorption*, in which the thrombus is gradually softened and absorbed, the vessel becoming pervious again. A clot of small or moderate size, good general health, and an abundance of blood vessels in the neighborhood of the thrombus are necessary for resolution to occur.

3. *Organization*—a frequent change—in which connective tissue gradually takes the place of the thrombus. White blood corpuscles enter the thrombus from the wall of the vessel and small blood vessels from the same source, the corpuscles leading to the formation of the connective tissue.

4. *Calcification*, or the formation of a mass of chalk. Its production will be explained in connection with calcareous degeneration.

5. Softening, which may be

(1) *simple*, from the gradual breaking down of the clot into a mixture of granular and fluid matter looking like pus ;

(2) *infective*, in which the softened, broken up thrombus contains germs which render it infective, as in the thrombotic form of puerperal septicæmia.

6. *Putrefaction* due to the entrance into the clot of the germs of decomposition.

Results of Thrombosis.—1. *Changes in the wall of the vessel* at the seat of the thrombus, due to the thrombus acting as a foreign body and setting up inflammation and causing thereby

(1) cloudy swelling and desquamation of endothelium ;

(2) infiltration of the walls of the vessel with serum and leucocytes ;

(3) later on thickening and hardening of the wall from the formation of new connective tissue.

2. *Obstruction to the circulation.*

3. *Embolism*, from lodgment of a piece of the clot in some other vessel.

EMBOLISM.

Definition.—The plugging of a vessel by a body, usually solid, which was circulating in the blood and lodged in a vessel too small for it to pass through.

Sources of Emboli.—1. *Thrombi.*

2. *Vegetations, atheromatous, or calcareous masses* from the cardiac valves.

3. *Pieces of new growth*, as cancer or sarcoma . . .

4. *Parasites*, little clumps of which sometimes gain access to the vessels in certain acute infectious diseases, as dysentery.

5. *Fat*, particles of which sometimes gain access to the vessels especially in cases of fracture of bones.

6. *Pigmentary matters*, a rare source

7. *Air*, a very unusual source of emboli.

Emboli found in the *pulmonary* artery or its branches or the pulmonary capillaries, usually come from the veins or the *right* side of the heart.

Emboli found elsewhere in the body *except in the liver* usually come from the arteries or the *left* side of the heart.

Emboli found in the *liver* usually come from the organs connected with the *portal system* of veins.

Results of Infective Embolism.—1. *Infective inflammation and abscess* at the seat of lodgment from the action of germs and the ptomaines formed by them.

Results of Simple Embolism.—1. *Irritation, inflammation and softening* of the wall of the vessel at the seat of lodgment, with subsequent formation of an aneurism there.

2. Local anæmia and its results.

3. Infarctions.

INFARCTIONS.

Seats.—Any organ or part having terminal vessels.

Shape.—Pyramidal from arrangement of the branches of the artery.

Varieties.—1. *White*, from coagulative necrosis of tissues deprived of blood.

2. *Red*, from the passage of blood from neighboring capillaries into those whose supplying artery is plugged. The red corpuscles and other elements of the blood then pass out of these vessels and collect in the tissues around.

Terminations.—1. Absorption of the greater part of the exudate and necrosed mass and formation of a cicatrix,

2. Partial liquefaction and cyst formation.

3. Calcareous degeneration.

4. Gangrene from entrance of germs of decomposition.

PATHOLOGICAL RELATIONS OF BLOOD PRESSURE.

Hydro-static pressure influences the venous system only and is of practical importance in connection with varicose veins of the lower extremities.

Hydro-dynamic pressure or arterial tension is dependent on:

1. The *volume* of blood.

2. The *size of the vessels*; for the amount of blood being the same the greater the capacity of the arterial system the less will be the tension.

3. The *facility with which the arteries empty into the capillaries*; facility of discharge of blood of course lessens vascular tension in the arteries.

4. The *force of the heart*; it is manifest that other things being equal the blood pressure will be in direct proportion to the strength of the heart's beat.

Causes of High Arterial Tension.—1. *Increased heart power* from exertion, stimulants and certain diseases, as hypertrophy.

2. *Increased resistance of the vascular walls* from spasm of the muscular coat or atheromatous disease, or from arterio-capillary fibrosis.

3. *Increased resistance* accompanying cirrhotic Bright's disease.

Evidences of High Tension.—1. *Incompressibility* of the *arteries*, because an elastic tube *filled* with fluid is much less compressible than one partially filled.

2. *Accentuation* of the *aortic second sound* because the tension of the vessels forces the blood back against the aortic valves at the end of the systole and causes them to shut with unusual force, thus causing an accentuation of the second sound.

3. *Prolonged first sound* of the heart, because the heart cannot drive the blood as rapidly into *full* vessels as into those which are less full.

4. *Abundant flow of limpid urine*, because the amount of water discharged by the kidneys is in direct proportion to the blood pressure.

Results of High Tension.—1. *Cardiac* or *præcordial* pain the cause of which is not clear.

2. *Atheroma* of the *vessels*.

3. *Apoplexy*, in consequence of the atheromatous disease of the arterial walls and the increased pressure of the blood in the vessels.

Causes of Low Arterial Tension.—1. *Diminished cardiac force* brought about by

- (1.) direct or reflex nervous influence;
- (2.) exhausting diseases or loss of blood;
- (3.) certain diseases of the walls of the heart;
- (4.) certain drugs;

2. *Diminished peripheral resistance* due to

- (1) exhausting diseases;
- (2.) certain drugs, as aconite and amyl nitrite;
- (3.) nervous influences as in shock, when the vessels of the splanchnic area are greatly dilated and the tension in the arteries correspondingly diminished.

Evidences of Low Arterial Tension.—1. *A feeble and short first sound* of the heart because in consequence of the dilatation of

the vessels but little blood passes into the ventricle during diastole, and as there is but little pressure in the vessels, a feeble contraction of the ventricle is sufficient to empty it.

2. A *compressible pulse*, because the vessel walls are not put upon the stretch from internal pressure.

3. A *scanty flow of urine* for obvious reasons.

4. *Pallor and faintness* because the blood is chiefly in the dilated vessels of the splanchnic area and the skin and cerebral centres are badly supplied with blood.

INFLAMMATION.

Definition.—The series of changes which occur in the tissues when an irritant is applied, provided such irritant is not sufficient to cause the death of the tissues.

The Changes in the Blood Vessels and Circulation are—

1. Sometimes a *fleeting contraction* of the smaller vessels, which is due to the direct effect of the irritant upon the contractile muscular tissue or the contractile protoplasm.

2. *Dilatation* of the small vessels, which always occurs and may be due to one of three things—

(1) paralysis of the vaso-constrictor nerves ;

(2) irritation of the vaso-dilator nerves ;

(3) paralysis and loss of tone of the *walls* of the vessels themselves.

A change in the vascular *wall itself* is the probable cause of dilatation of the vessels.

3. *Retardation* of the blood current.

4. *Sticking* of *white corpuscles* to the sides of the *small veins* and gradual blocking of the vessels thereby.

5. *Impaction* of the *capillaries* with *red* blood corpuscles, oscillation, stasis and thrombosis.

6. *Escape of fluid* and both *white* and *red* corpuscles from the vessels.

All of these phenomena are due to a change in the vascular wall, which leads to stagnation and passive hyperæmia, and these cause a still further change in the vessel wall by which it becomes more permeable to fluids and also to the blood corpuscles. The white corpuscles pass out by *diapedesis*—a little off shoot passing through the wall first and the rest of the corpuscle following it; the *white* corpuscles are *active* and not passive agents in this process. The red blood corpuscles and fluid are forced out by pressure, but pressure would be insufficient to produce such a result unless there had been a change in the vascular wall making it more permeable. The white corpuscles pass out of the small

veins chiefly, the red pass out of the capillaries; neither of them pass through the *arterial* walls.

The Essential Lesion of Inflammation is a change in the vascular wall, as is shown by the following facts:

1. Inflammation will occur in a part when all the nerves going thereto have been divided.
2. The blood which was retarded while passing through an inflamed spot will flow freely after it passes it.
3. If milk be allowed to flow through a frog's vessels, instead of blood, it will be retarded in its course at an inflamed spot just as blood would be.
4. Irritant fluids passing through the vessels will set up inflammation.

The Changes in Inflamed Tissues are—1. *Infiltration* with fluid and white and red corpuscles, which has just been explained.

2. *Cloudy swelling* of the tissue cells, which is a *degenerative* change and is attended by a loss or diminution of function.

It was formerly thought that there was a proliferation of the connective tissue corpuscles in inflammation, but this view has been abandoned.

The Probable Functions of the White Corpuscles which pass out of the vessels in inflammation, are—1. To act as scavengers and to carry away waste products, such as broken up red and white corpuscles, parasites, &c.

2. To destroy parasites (bacteria) by surrounding them, each cell taking up one or more parasites, and then destroying them by a process of digestion. Such cells or leucocytes are called *phagocytes*.

The Ultimate Disposition of the Leucocytes which pass out of the vessels in inflammation.

1. Some of these leucocytes probably return to the circulation by the lymph channels immediately.
2. Some take up into their substance the products of the inflammation and carry them off through the lymph channels and thence into the blood vessels, which in turn carry the effete matters to the eliminative organs.
3. Some take up bacteria into their substance and either kill the bacteria or are killed by them. If they kill the bacteria they carry them off just as they would any other effete matter; if the bacteria kill the cells they either break up and are carried off by living cells, or if pyogenic germs are present pus is formed which may be (1) absorbed, (2) evacuated by the bursting of the abscess, or (3) may undergo some form of degeneration.
4. Finally, such cells may be converted into *connective tissue* or into *giant cells*. (See Nature of the Exudate.)

Explanation of the Clinical Phenomena of Inflammation.—

1. *Redness* is due to the dilatation of the vessels and the increased amount of blood in the tissues therefrom.

2. *Heat* is due to the same cause.

3. *Swelling* is due to the exudation from the blood vessels into the tissues.

4. *Pain* is due to (1) the pressure of the exudate on the terminal filaments of the sensory nerves and (2) to the irritation of such filaments by the leucomaines formed by the germs.

5. *Fever* is due to an effect produced on the heat regulating apparatus by (1) reflex action or (2) the absorption of leucomaines and their action on the heat centre or centres.

6. *Defective nutrition* is due to (1) the defective supply of pure blood to the tissues and (2) probably also to an obscure condition of the tissues themselves which renders them unfit to absorb nutriment.

Nature of the Exudate in Inflammation.—The nature of the exudate varies in different cases according to :

1. The *intensity* of the cause.

2. The *duration* of the cause.

3. The *nature* of the cause.

4. The *resisting power* and the blood supply of the tissues.

The following are the different forms of exudate :

1. *Serous* or liquid, which occurs usually in inflammations which are mild in intensity. It contains leucocytes in small numbers.

2. *Fibrinous* in which the exudate soon becomes firm or "set ;" inflammations in which this form of exudate occurs are usually acute in duration and of considerable intensity. A greater or less number of leucocytes is found caught in the fibrinous exudate.

3. *Productive* in which a large number of cells pass into the tissues, with little intercellular substance, and these cells may undergo one of two changes ; (1) they may grow and elongate and then form connective tissue or (2) several cells may unite to form a very large cell which is branched and which is called a giant cell. On the surface of a healing wound there often appear little elevations or "granulations" each of which has in its centre a little capillary loop and around this loop a number of cells some of which are ordinary leucocytes and some have grown, so as to form much larger oval cells with a very distinct nucleus ; such enlarged cells usually form connective tissue (a scar) and are called fibro-blasts or formative cells or (epithelioid) cells. They have no connection whatever, however, with epithelium.

4. *Suppurative*, in which the exudate consists of a great number of cells with a liquid intercellular substance. The cells are called pus cells ; they are white corpuscles which have passed out of the vessels and have died either from want of nourishment or from the action of bacteria or their products (leucomaines). They

differ from living leucocytes in being more granular and containing three small nuclei, as a rule.

The Relation of Bacteria to Pus Formation.—The presence of certain bacteria is necessary to pus formation; the most important of these bacteria are (1) the staphylococcus pyogenes aureus, (2) the staphylococcus pyogenes albus, (3) the streptococcus pyogenes. These bacteria seem to have the power of killing many leucocytes and also of peptonizing and liquefying the exudate, which would otherwise be fibrinous.

6. Hemorrhagic, in which great numbers of red corpuscles pass out of the vessels as well as white. This can only occur when the nutrition of the vascular walls is very seriously impaired, and hence only occurs in cases where the inflammation is severe in intensity.

The Causes of Inflammation.—A. The *predisposing* cause, which is often, but not necessarily, present, is defective nutrition of the tissues which may be

1. Constitutional, or
2. Local.

Constitutional defects in the strength and vigor of the tissues may be

- (1) *hereditary*, as in scrofula and phthisis; or
- (2) *acquired*, as in alcoholism, diabetes, Bright's disease, &c.

Local defects in the nutrition of a part are due usually either to *injury* or *local* anæmia; some defect of development may cause it.

B. The *exciting* causes of inflammation may be—

1. Traumatic.
2. Trophic. (?)
3. Bacterial or infective.

1. The *trauma* or injury in the traumatic form of inflammation may be produced by (1) mechanical, or (2) chemical violence or (3) by the action of heat or cold.

The Characteristics of Inflammation Due to a Trauma are—

- (1) it shows no tendency to spread;
- (2) it has no tendency to continue when the cause is removed.
2. The *trophic* disturbances of nutrition are due to nervous influence. The affection of the nerve leading to such an inflammation may be situated either in the nerve centers or in the nerve fibres.

It is not certain that trophic disturbances are exciting causes of inflammation; they may merely interfere with the nutrition of a part and hence render it more liable to inflammation when an exciting cause arises.

3. *Bacteria*, fungi or germs are by far the commonest causes of inflammation. They may act either

- (1) *directly* upon the tissues, or

(2) *indirectly* by the formation of leucomaines.

The Characteristics of Inflammation due to Bacteria are—

1. It is apt to spread.
2. It is usually more tedious than an inflammation due to trauma.

The modes in which such an inflammation may spread are—

1. By *continuity* or *contiguity* of tissue, as in simple abscess.
2. By the *lymphatics*, as in lymphadenitis from an infected wound.
3. By the *blood vessels*, as in the formation of “meta-static” abscesses from septic emboli.

The Circumstances Which Influence the Action of Germs in Producing Inflammation are—

1. *Arrest of the bacteria*, as in septic emboli and lymphadenitis.
2. *Predisposition* on the part of the tissues.
3. The *seat of inoculation* and the anatomical arrangement of a part.
4. The *blood state*, such as that of diabetes in which abscesses and carbuncles are very apt to occur.
5. The *species of germ*; for instance, streptococcus pyogenes is associated with *diffused* suppuration and the staphylococcus pyogenes aureus with *localized* suppuration.
6. The *number* of germs introduced at a time.
7. The *virulence* of the germs or the leucomaines generated by them; virulence is lessened by various circumstances.
8. The *presence of other species* of germs; some increasing the virulence of each other and some being mutually antagonistic.
9. *Probably the season* of the year or atmospheric conditions.

The Modes of Arrest of Inflammation.—1. When the inflammation is *traumatic* in origin it ceases so soon as the trauma is removed and the exudate is in part removed by the white blood cells and in part becomes organized.

2. When the inflammation is *bacterial* in origin leucocytes pass out of the vessels so as to form a wall around the seat of the bacteria and there seems to be a sort of battle between the two (leucocytes and bacteria).

Some of the leucocytes are destroyed by the *leucomaines* generated by the bacteria.

The *bacteria* are destroyed by (1) the leucocytes enveloping and digesting them, and (2) by the leucomaines which they have themselves generated.

The Varieties of Inflammation with Respect to the General symptoms are—1. *Sthenic*, which is characterized by a full, strong pulse, flushed face, and usually by excitement.

2. *Asthenic*, characterized by exhaustion.

3. *Typhoidal*, in which there is not only great prostration, but a dry, brown tongue, sordes on the teeth, low muttering delirium and diarrhœa.

The Varieties of Inflammation Classified with Reference to the part involved and the character and nature of the exudate are—1. *Catarrhal*, in which the mucous membranes are affected. The exudate is serous in character, but contains large quantities of mucous and great numbers of epithelial cells which have desquamated and undergone fatty degeneration; there is also a considerable number of leucocytes.

2. *Croupous and diphtheritic*, in which mucous membranes are affected and the exudate is fibrinous in character. A *croupous membrane* is chiefly on the *surface* of the mucous membrane and consists of fibrin, leucocytes and superficial flakes of mucous membrane which have undergone coagulative necrosis.

A *diphtheritic* membrane affects the mucous membrane more *deeply* and seriously than a croupous one; it consists of fibrin, leucocytes, thick masses of mucous membrane which have undergone coagulation necrosis, and also great numbers of micrococci.

3. A *parenchymatous* inflammation is one in which the *essential* cells of an organ are chiefly involved.

4. In *interstitial* inflammation the connective tissue frame work of an organ is chiefly affected.

Acute and Chronic Inflammation.—1. *Acute* inflammation runs a rapid course; it may be mild or severe in intensity.

The exudate may be serous, fibrinous, purulent or hemorrhagic. The vessels have lost the *power* to contract.

2. *Chronic* inflammation runs a slow course, and is usually comparatively mild in intensity.

The exudate may be serous, fibrinous, purulent, or productive. The blood vessels have the power to contract, but must be stimulated to do so.

Terminations of Inflammation.—1. *Resolution*, in which the exudate is absorbed and the tissues return to their normal condition; the requisite conditions are (1) removal of the cause, (2) return of the vessel wall to a healthy condition, (3) absorption of the exudate.

2. *Organization*, in which connective tissue is formed from the leucocytes.

3. *Styting and discharge* (abscess formation).

4. *Certain degenerations*, such as fatty and calcareous.

5. *Necrosis* or death of the tissue from either (1) the direct action of the irritant upon the tissues, or (2) the pressure of the exudate on the blood vessel and the consequent cutting off of blood.

Regeneration and Repair of—1. *Nervous, muscular, glandular, and epithelial* tissues.

Nerve ganglion cells are *never* regenerated.

Nerve fibres and muscular fibres may be regenerated by growth and division of their nuclei.

Glandular and epithelial tissues can only be regenerated by the multiplication of cells of the same character as the tissue itself.

None of these tissues are regenerated from leucocytes.

2. *Connective tissue* is regenerated by (1) the conversion of leucocytes into connective tissue fibres or cells, and (2) the multiplication of connective tissue corpuscles.

The Conditions Favorable for Regeneration and Repair are:

1. The removal of the cause.
2. An abundant supply of pure blood, and
3. In the case of nervous and muscular fibres and glandular and epithelial tissues the presence of such tissue at the inflamed spot.

The Treatment of Inflammation.—The indications are—

1. To lessen the amount of blood in the inflamed part.
2. To relieve pain.
3. To lessen or relieve fever.
4. To promote resolution.
5. To destroy bacteria, or prevent the effects usually produced by them.

1. To lessen the amount of blood in the inflamed area in *acute* cases.

(1) *blood-letting*, general or local, has been used; it is of comparatively little value;

(2) Agents which *dilate the vessels of the skin*, and thus produce a collateral anæmia of internal organs, are extremely useful. Such agents are *poultices* and *diaphoretics*, and also nauseants, aconite and veratrum.

To lessen the amount of blood in an inflamed part in *chronic* cases, agents are indicated which will stimulate the walls of the vessels to contract, such as

- (1) *counter-irritants*;
- (2) *ergot*;
- (3) *electricity*;
- (4) *massage*;
- (5) *astringents*;

2. To relieve pain:

- (1) *opium* and other *analgesics*, such as phenacetine, antipyrine, acetanilide, exalgine, &c.;
- (2) *heat*, which relaxes the tissues and relieves tension;
- (3) *cold*, which lessens the power of sensory fibres to convey painful impressions;

(4) *local blood-letting*, which relieves the tension in the tissues (pressure) directly.

3. To lessen or relieve fever—see “Fever.”

4. To promote resolution, suitable nourishment and tonics are indicated in order to furnish proper blood for the repair of the vascular walls and other tissues.

5. To destroy bacteria and prevent their effects, *antiseptic treatment* is to be employed, as the use of antiseptic sprays, as in diphtheria.

FEVER.

Animal Heat is due to oxidation of *food* usually, but probably of the *tissues* also, in disease.

The oxidation takes place in the muscles and glands, especially the former.

Influence of the Nervous System on the Production of Heat. There are two sets of nervous influences which regulate the *production* of heat—(1) *thermogenic*, or heat forming, and (2) *thermo-inhibitory*, which check heat formation.

Thermogenic centres exist apparently both in the brain and spinal cord.

Thermo-inhibitory centres exist in the brain only.

The regulation of the body heat is called *thermo taxis*.

About 80 per cent. of the heat loss occurs from the skin by

(1) radiation of heat;

(2) evaporation of sweat.

The greater the amount of blood in the skin the greater will be the heat loss, and the amount of blood in the skin is regulated by (1) the size (dilatation) of the vessels and (2) the force and frequency of the heart's action.

About 20 per cent. of the heat loss occurs from the lungs, the heat being expended in warming the inspired air. So that the more rapid the respirations the greater will be the heat loss.

The Normal Temperature of the Human Body is:

1. In the axilla from 98° – 99° .

2. In the mouth from 98.5° – 99.5° .

3. In the rectum from 99° – 100° .

There is a *diurnal* variation in health, the temperature being about three-fourths of a degree higher in the afternoon from 4 to 6 than it is in the early morning hours from 2 to 4. The temperature of the body remains practically constant in winter and summer, and is regulated in the following manner:

1. *Heat*, whether from without or as a result of internal combustion (violent exercise, &c.,) causes:

(1) *dilatation* of the *vessels* of the skin and loss of heat by radiation.

(2) *sweating* and loss of heat by evaporation;

(3) more *rapid breathing* and consequent increased loss of heat in warming the inspired air.

2. *Cold* causes:

(1) *contraction* of vessels and less radiation;

(2) *dryness* of the skin and less evaporation, and also less radiation because a moist skin is a better conductor than a dry one;

(3) *slower respiration*, so that less air is taken in inspiration and less heat is consequently expended.

The Nature of Fever.—Fever is caused by *excessive heat formation* and *not* by diminished heat loss; this is proved by the following facts:

1. The amount of CO_2 and urea is *increased* in fever.

2. If a fever patient and a well person are placed in water of the same temperature, the fever patient will heat the water more than the well person.

The Phenomena of Fever and Their Explanation.—1. Disturbances of temperature.

(1) the temperature is *elevated*: 101° . Fahr. is a slight fever; 102.5 a moderate fever and a temperature over 103° . a high fever; a temperature of 106° . is very serious, and recovery is rare if the temperature reaches 110° ;

(2) the diurnal variation is usually observed as in health;

(3) the temperature is more easily affected in fever than in health; some antipyritics will reduce the temperature in fever and not in health.

The disturbances of temperature are due probably to some defect in the action of the thermo-genic or thermo-inhibitory heat centres, and this defect may be caused by:

(1) the action on the centres of *certain morbid products* (leucomaines) circulating in the blood as in scarlet fever, for instance;

(2) *reflex action* as in the so-called "urethral fever", observed sometimes after passing a bougie;

(3) some *functional disturbance* of these centres as in certain cases of hysteria with high temperature;

(4) some *injury of the heat centre* or conducting fibres.

2. Disturbances of the *circulatory system*:

(1) the *pulse* is more rapid because the heart is stimulated by *warm blood*. In the late stages of fever the pulse becomes weak in consequence of albuminoid degeneration of the muscular tissue of the heart (from leucomaines);

(2) the *red corpuscles* are *diminished* in number, because the food is burnt off and the normal formation of corpuscles is prevented.

3. Disturbances of the *respiratory system* :
 - (1) the respirations are more frequent because the over-heated blood stimulates the respiratory centre, and *possibly* leucomaines have the same effect ;
 - (2) more CO_2 is discharged because of increased combustion.
4. Disturbances of the *nervous system*, excitement, delirium, stupor, &c., due to the action of *over-heated blood* and *leucomaines*,
5. Disturbances of the *muscular system*, such as weakness and twitching of the muscles from
 - (1) degenerative changes due to leucomaines.
 - (2) actual consumption of muscular tissue.
6. Disturbances of the *digestive system*, such as loss of appetite, nausea, constipation, &c., due to
 - (1) albumoid degeneration of secretory and absorbant cells caused by leucomaines ;
 - (2) degenerative changes in the muscular coat of the bowels produced in the same way.
7. Disturbances of the *excretory organs* (kidneys, &c.)
 - (1) less water discharged than in health, because the blood pressure is lessened ;
 - (2) more urea and uric acid are discharged in consequence of increased oxidation ;
 - (3) sometimes albuminuria occurs from the degenerative changes in the cells of the urinary tubules, such changes being caused by leucomaines.
8. Disturbances of nutrition (emaciation and debility) in consequence of
 - (1) improper quality of the blood ;
 - (2) impaired action of the digestive and absorbent glands ;
 - (3) direct destruction of tissue by oxidation, in the febrile process.

The Modes of Termination of Fever are—1. *Crisis*, when the temperature falls suddenly.

2. *Lysis*, when there is a *gradual* fall of temperature.

The Varieties of Fever classified according to the course of progress of the symptoms, especially the elevation of temperature, are—1. *Continued*, when the temperature remains elevated with only a slight diurnal variation.

2. *Remittent*, when the temperature undergoes marked remissions.

3. *Intermittent*, when the patient is at times free from fever ; in such cases the fever usually recurs periodically.

4. *Relapsing*, in which there is apparent recovery and then a relapse.

Classification According to the Severity and combination of

the symptoms.—1. *Simple*, in which the rise of temperature is not accompanied by any other serious disturbances.

2. *Hectic* or *suppurative*, which is caused by the absorption of leucomaines generated in suppuration.

3. *Adynamic*, in which there is great depression of the vital powers.

4. *Typhoidal*, in which there is prostration, dry and brown tongue, low muttering delirium, sordes on the teeth and usually diarrhœa

5. *Malignant*, in which a large quantity of poisonous leucomaines is suddenly thrown into the system and overwhelms the patient.

The Causes of fever have already been stated under the head of "Disturbances of Temperature."

The Prognosis of Fever is Dependent on—1. Its intensity and duration.

2. Its type, whether simple, typhoidal, &c.

3. The previous health of the patient, a person in impaired health succumbing more readily to a febrile attack.

4. The existence of complications, which nearly always render the prognosis worse.

The Treatment of Fever.—The *indications* are—1. To lessen heat production.

2. To increase heat loss.

The agents which *lessen heat production* are—

- (1) antipyrine ;
- (2) acetanilide ;
- (3) phenacetine ;
- (4) salicylic acid and its salts ;
- (5) alcohol, to a slight extent.

The agents which increase heat loss are—

1. Such as *increase the dilatation of the vessels of the skin*, and
2. *Increase the amount of sweat.*

The same drugs for the most part act in both ways ; these drugs are—

- (1) aconite ;
- (2) veratrum ;
- (3) sweet spirits of nitre ;
- (4) acetate of ammonia, &c. ;
- (5) opium, especially Dover's powder.
3. Agents which *withdraw heat directly*, such as
 - (1) the cold bath ;
 - (2) cold sponging ;
 - (3) the wet pack.

Diet in Fever.—The diet in fever cases should be *easily digest-*

ed and assimilated, because of the condition of the digestive glands, and *nutritious*, because of the waste which fever causes.

ATROPHY.

Definition.—A wasting or diminution in size of a part or organ.

The Varieties of Atrophy are—1. *General*, when the whole body is involved.

2. *Local*, when the atrophic change is localized.

The Changes in an Atrophied Organ, as a rule, are—1. *Diminution* in the size or number (or both) of its essential elements (such as cells or nerve or muscular fibres), and

2. An *increase* in the connective tissue frame work.

The Causes of (A) General Atrophy are—1. *Defective nourishment* as in cases of cancer of stomach or atrophy of the gastric tubules.

2. *Excessive waste*, as in consumption and profuse suppuration and also in chronic diarrhœa.

3. *Impaired vitality*, as in old age when the cells are incapable of appropriating nourishment in proper quantity.

The Causes of (B) Local Atrophy. are—1. *Defective supply of blood* to a part from narrowing of a vessel, or a partial cutting off of the blood supply.

2. *Diminished functional activity*, as the atrophy of muscles which occurs from prolonged disuse.

3. *Defective nervous influence* (tropho-neuroses), as in infantile paralysis (or acute anterior polio-myelitis.)

HYPERTROPHY.

Definition.—An increase in the size of a part or organ.

The Varieties of Hypertrophy are—1. *Simple* when there is an increase in the *size* of the cells of which a tissue is composed without any increase in number, and

2. *Hyperplasia* when there is an increase in the number of the cells.

As a rule, the cells are increased both in size and number.

The Causes of Hypertrophy are—1. *An increased blood supply*, as in acne rosacea.

2. *The eased functional activity*, which is the cause of the enormous development (hypertrophy) of certain muscles from use, such as hypertrophy of the heart.

The enlargement of an organ or part which occurs as a result

of inflammation is not a *true* hypertrophy, because the enlargement is due to the formation of connective tissue.

THE DEGENERATIONS.

Definition.—By the degeneration of a tissue or organ is meant such a change in its quality that it is rendered less capable of performing its functions.

PARENCHYMATOUS DEGENERATION.

Definition.—A change in the tissues, usually the parenchyma, of an organ by which albuminous granules appear in its cells.

Synonyms.—Albuminoid degeneration; granular degeneration; cloudy swelling.

Nature and Appearance.—Tissues in which such a degenerative change has occurred contain granular, albuminous particles which are soluble in acetic acid. The tissue cells are opaque and swollen.

The Seats of cloudy swelling are—1. The *cells* of *muscular tissue*, such as that of the heart, &c.

2. The *cells* of *glandular organs*, as the liver and kidneys.

3. *Connective tissue* cells, as in inflammation of the cornea.

The Causes of cloudy swelling are—1. Possibly a prolonged high temperature.

2. The action of leucomaines.

3. Certain poisons, such as arsenic and phosphorus.

Cloudy swelling is always a step towards *death* of the tissue.

The Results of Albuminoid Degeneration are—1. Impaired function.

2. Usually a return to the normal state, but

3. It may result in fatty degeneration.

MUCOID AND COLLOID DEGENERATION.

Definition, Nature and Appearance.—1. In *mucoïd* degeneration a substance is formed resembling mucous in appearance.

2. In *colloid* degeneration a jelly-like mass is formed.

Colloid matter differs from mucoïd furthermore in containing sulphur, and not responding to the tests for mucine.

The Seats of (A) Mucoïd degeneration are—1. The epithelial cells lining the mucous membranes in catarrhal inflammations.

2. Certain new formations, such as the myxo-sarcomata, occasionally the connective tissue, as in myxœdema.

The Seats of (B) Colloid degeneration are—1. Certain tumors, especially malignant growths of the ovaries.

2. Occasionally cartilage.

The *causes* of mucoid and colloid degeneration are unknown.

The *results* are loss of function, and the tissues affected never return to their normal state.

FATTY DEGENERATION.

The Varieties of Fatty Degeneration are—1. Fatty *infiltration*, when the fat is deposited between the fibres of muscles or in a gland *without* destruction of its protoplasm, and

2. Fatty *metamorphosis* when the fat takes the place of the protoplasm of the cell or muscle fibre.

In the case of glands, such as the liver, it is sometimes difficult to distinguish fatty infiltration from fatty metamorphosis.

The Nature and Appearance of tissues which have undergone fatty degeneration. They are—1. *Pale*, because the blood vessels are compressed.

2. *Yellowish*, from the presence of fat.

3. *Softened*, for the same reason.

The Chief Seat of Fatty Infiltration is in the connective tissue; and it is of serious moment when situated in the connective tissue, between the muscular fibres of the heart; it is common also in the liver cells.

The Chief Seats of Fatty Metamorphosis are—1. *Muscular tissue*, as in certain cardiac troubles.

2. The *cells* of *glandular organs*, as in certain cases of Bright's disease.

3. The *exudation* of *inflammation*, especially when rich in cells.

4. The *coats* of the *small arteries*.

The Causes of fatty degeneration are—1. The excessive use of *fatty food*, or food from which fat is formed.

2. A *sedentary* and indoor life.

3. *Defective oxidation* from either general or local anæmia.

4. *Alcohol*, which probably interferes with oxidation or else is burnt off in place of fat in heat formation.

5. Certain protoplasm *poisons*, such as phosphorus.

6. *Lowered vitality* from any cause, such as old age, &c.

The Results of fatty degeneration depend upon the organ affected.

The Termination is cheesy degeneration from the drying up and crumbling of the cells which have undergone fatty degeneration, and this caseous mass may undergo either

1. *Softening*, if so situated that fluid can get access to it, thus forming a milky-looking fluid (pathological milk) or

2. *Calcareous* degeneration, q. v.

Cheesy or caseous degeneration, as it is sometimes called, is especially apt to occur in lymphatic glands, and such masses may remain there for sometime before undergoing softening or calcification.

CALCAREOUS DEGENERATION.

Definition.—A deposit of calcareous matter in certain tissues or parts.

Synonym.—Calcification, ossification (of the heart or arteries).

The Nature and Appearance of a part in calcareous degeneration. There is a deposit of lime-salts which renders the part hard, brittle, and granular. Rarely there is actual formation of bone.

The Usual Seats of calcareous degeneration are—1. *Inflammatory exudates*, as in some cases of peri-carditis and of pulmonary phthisis.

2. *Tumors*, such as uterine fibroids.

3. The *coats of arteries*, as in atheroma or calcification; these changes in the arteries frequently lead to thrombosis and embolism and to attacks of angina pectoris.

The Causes of calcification are—1. *Previous disease* of the tissues by which their vitality is depressed.

2. *Old age*, which probably causes a depression of the vitality of the tissues.

3. *Fatty degeneration*.

The *immediate* or *actual* cause of calcification is not known. The above are only predisposing causes.

The Results and Terminations depend on the organs or tissues involved.

AMYLOID OR WAXY DEGENERATION.

Nature and Appearance. Tissues which have undergone this degeneration have a waxy appearance, the cells become translucent and merge into each other so that their outlines are lost; the organ is firm and brittle at the degenerated point. If tincture of iodine be poured over the seat of waxy degeneration it will produce a mahogany color.

The nature of the degeneration is unknown.

The Usual Seats of the degeneration are—1. The spleen.

2. The liver.

3. The kidneys.

4. The bowels.

5. The muscles.

The change begins in the wall of the blood vessels.

The Causes of waxy degeneration are—1. *Prolonged suppuration*, especially from disease of the bones.

2. *Syphilis*.

3. *Possibly cancer, gout, &c.*

The Result is permanent loss of function.

The Termination is never in recovery so far as known and sooner or later such cases end in death.

FIBROID DEGENERATION.

Definition.—A change in an organ or part, consisting in an atrophy of its parenchyma and an increase in its connective tissue frame work.

Synonyms—Fibroid substitution.

Nature and Appearance.—In fibroid degeneration the organ or part is usually smaller and harder than normal.

The change may occur in three ways: 1. There may be first atrophy of the parenchyma, and then an increase of connective tissue in consequence.

2. The increase of connective tissue may be the primary change and may cause atrophy by pressure on the parenchymatous cells and their nutrient vessels.

3. The two processes may be simultaneous and due to the same cause.

As a general rule, fibroid degeneration is the result of chronic (productive) inflammation, but it is possible in certain cases (systemic cerebral and spinal lesions) that it is not inflammatory.

The Usual Seats are—1. The *liver* (in cirrhosis).

2. The *kidney* (in the sclerotic form of Bright's disease).

3. The *blood vessels* (in arterio-capillary fibrosis).

4. The *nerve centres* (in systemic lesions).

The Causes differ in different cases, and will be studied in connection with individual diseases.

The Results depend on the seat.

The Termination is in permanent loss of function.

TUBERCLE AND TUBERCULOSIS.

Definition.—By tubercle is meant a small enlargement, inflammatory in character, consisting usually of one or more giant cells, surrounded by epithelioid cells and leucocytes, and having a reticulum or stroma of homogeneous or fibrillated tissue. Tubercles are due to inflammatory action, set up by the "*bacillus tuberculosis*;" and the infectious disease "*tuberculosis*" is due to the tubercle bacilli.

Varieties of Tubercle.—1. *Grey*, when *fresh*, and fatty degeneration of the cells has not occurred.

2. *Yellow*, formed from the grey by fatty degeneration and caseation of the cells.

Size of Tubercle.—A single tubercle is about $\frac{1}{12}$ of an inch in diameter, but a number may be united to form a mass as large as a hazel nut, or much larger.

Seats of Tubercle.—1. *Respiratory* organs or tracts.

2. *Serous membranes*, such as the pleura, peritoneum, cerebral meninges, joints, &c.

3. *Mucous membranes* of bowels, &c.

4. *Lymph glands, kidneys, spleen, testicles, bowels, skin*, &c.

When Most Liable to Occur.—Tubercle is most liable to occur in childhood or early adult life, but no age is exempt.

Histological Structure.—A tubercle is usually formed of the following parts—1. One or more giant cells in the centre.

2. A number of epithelioid cells around these giant cells.

3. A still larger number of leucocytes around the epithelioid cells.

4. A delicate reticulum or stroma which may be homogeneous or fibrillated.

No new blood vessels are ever found in tubercle.

In cases of acute tuberculosis lymphoid cells (or leucocytes) predominate, and may be the only ones present.

Origin of Cells in Tubercles.—Tubercle is an inflammatory formation, and the cells in it are derived from—1. White blood cells.

2. *Possibly* from epithelium, but this is extremely doubtful.

Secondary Changes in Tubercle.—The secondary changes are—1. *Caseation*, which is by far the most common, and

2. *Fibroid* change—the formation of connective tissue.

Caseation occurs especially in the glands, but it often occurs in other places; the caseous change commences in the centre of the tuberculous mass.

Fibroid change occurs on serous membranes and in chronic

cases of pulmonary tuberculosis. Frequently fibroid change occurs around a caseous mass, and thus serves as a protecting wall.

The Further Changes after Caseation are—1. *Encapsulation* when the outer layer of leucocytes becomes converted into connective tissue and forms a wall around the caseous mass.

2. *Calcification*, from a deposit of lime salts in the caseous formation. This occurs most frequently in the mesenteric glands, but is not uncommon in the lungs and other organs.

3. *Softening*, and the formation of cavities in the lungs or of cold abscesses in connection with caseous bones, &c.

The Terminations of Tuberculosis.—1. The tuberculous matter may be *discharged* or *removed artificially*, and the resulting wound may heal by scar tissue.

3. "*Obsolescence*" may be induced by

(1) the formation of a wall of connective tissue around the tubercle, or

(2) the *calcification* of the caseous mass.

4. *Death* may be caused in a number of ways—

(1) by *poisoning*, as in acute tuberculosis ;

(2) by *exhaustion*, the usual mode, &c., &c.

As long as a caseous mass of tubercle remains in the body of a patient, even though it may be surrounded by connective tissue, such person is in constant danger of sudden and fatal extension of the tubercular disease.

Causes of Tuberculosis.—The *essential* cause is the *Bacillus Tuberculosis*.

The Characteristics of the bacillus tuberculosis are as follows : It is a single celled plant, rod shaped ; about $\frac{1}{12000}$ or $\frac{1}{8000}$ of an inch in length, and about one-sixth as broad as it is long. It has rounded ends and shows usually clear spots in its body. It is motionless. It probably forms spores.

The Conditions requisite for its development and growth are—1. It must be in an *animal body* ; either that of a human being or of some lower animal. It may *live* in the *dry* state outside of the body for several weeks or months, possibly, but is incapable of growth and development.

2. A *temperature* between 82° and 108° F. is necessary for its growth ; but it flourishes best between 98° and 102° ; it is quite resistant to both cold and heat ; but *prolonged* cold or heat will destroy the bacilli ; the spores have greater resisting powers than the bacilli.

3. A certain *predisposition* on the part of the person is necessary for the occurrence of tuberculosis. In what this predisposition consists is not known.

The Avenues by which the bacilli gain access to the body are—1. The *respiratory* passages; the dry bacilli constantly float in the air and are taken in in inspiration.

2. The *digestive organs*; the bacilli may be taken in with milk from tuberculous cows, or with the flesh of tuberculous animals, or flesh which has become infected secondarily (by flies, &c.)

3. By the *skin*, as in lupus and certain cases of chronic eczema, with consecutive disease of the lymphatic glands.

4. By *inoculation*, as in wounds; this is rare in the human subject.

The Action of the Bacilli in the Tissues.—The bacilli or the leucomaines generated by them act as irritants and cause inflammation just as any other irritant does, but their action is peculiar in two respects: (1) the mass of cells formed contains no blood vessel, and (2) caseation is very apt to occur.

The Modes in which Tubercle Spreads.—1. By *continuity* and contiguity of tissue, as in the lungs and bronchial tubes and in the pleura, &c.

2. By *passing along* the bronchial tubes, bowels, urinary passages, &c., from one point to another.

3. By *lymphatics*. This is a very common mode by which the disease spreads; a few germs only may get into the blood vessels in this way, or a softened gland may burst and pour a large number of bacilli into the thoracic duct and blood-current at once, thus leading to *acute tuberculosis*.

4. By the *veins* and *arteries*. The walls of both these sets of vessels may be perforated by tuberculous masses and bacilli may thus gain access to the circulation.

The Results of extension are the occurrence of tubercle in other parts of the body. If the number of bacilli suddenly thrown into the circulation is very large, *acute tuberculosis* will result from the involvement of many organs, and the quantity of poison (leucomaines) generated.

The Prognosis of chronic tuberculosis depends on many conditions, such as—1. The previous health and vigour of the patient.

2. The organ or organs involved.

3. The number of bacilli which gain access to the body.

4. The surroundings of the patient, with respect to dirt, &c.

The Treatment of Tuberculosis depends upon the organ involved in great measure. The chief indications are—1. To remove all caseous glands, &c.

2. To sustain the health and strength.

Koch's treatment is now (January 21st, 1891,) on trial, but as yet the results have not been satisfactory. His method consists in injecting into tuberculous patients a "lymph" composed of a glycerine extract of the bacilli tuberculosis.

ACUTE TUBERCULOSIS.

Definition.—An acute infectious disease due to the action of the bacillus tuberculosis.

Causes.—1. The sudden discharge of the contents of a caseous gland into the circulation.

2. Possibly the opening of a caseous tubercular mass in some organ into a blood vessel.

Often no cause can be discovered.

Morbid Anatomy.—1. *Miliary tubercles* in many organs of the body—the brain and the lungs are of especial moment, because they may cause special symptoms.

2. Old tubercular products (caseous or fibrous) are often found in some parts of the body.

Symptoms.—A. In the *generalized* form much like typhoid fever.

1. Temperature elevated from 100° to 105° from absorption of leucomaines.

2. *Respiration* is usually hurried and there is cough from bronchitis.

3. *Pulse* is rapid and weak from fever and exhaustion.

4. *Nervous* symptoms: delirium, stupor, &c., are due to leucomaine poisoning.

5. *Digestive symptoms*: Anorexia or vomiting sometimes occurs from the presence of leucomaines in the circulating blood; constipation sometimes occurs from muscular weakness, but *diarrhœa* is more common from accompanying intestinal tuberculosis.

6. *General*. Emaciation and exhaustion are marked in consequence of the fever.

B. In the *cerebral* form the meninges are chiefly involved. (See Basilar Meningitis.)

C. Occasionally the lungs are extensively diseased with corresponding symptoms. (See Pulmonary Phthisis.)

Physical Signs.—The physical signs depend upon the extent of the trouble in the different forms; in the generalized form they are very slight.

Diagnosis.—Distinguished from *typhoid fever* by the absence of rose spots, the rapid respiration, the absence of tympanites, and sometimes by the presence of *crackling* in the lungs.

Prognosis.—Extremely unfavorable; duration from a few days to eight or ten weeks.

Treatment.—1. To *sustain strength* by nutritious food and stimulants.

2. To *relieve symptoms*, such as diarrhœa, sleeplessness, delirium, &c., by appropriate remedies.

THE VEGETABLE PARASITES.

Classification.—All the vegetable parasites of pathological interest are *thallophytes*, or plants in which there is no distinction between the stem and the leaf.

Furthermore they are devoid of chlorophyl or coloring matter, and hence are *fungi* and not *algæ*.

Pathological Fungi.—The pathological fungi are of three kinds—1. *Bacteria* or *Shizomycetes* or *fission fungi*.

2. *Yeasts* or *Blastomycetes*.

3. *Moulds* or *Hyphomycetes*.

Of these the fission fungi or bacteria are by far the most important.

BACTERIA, OR FISSION FUNGI.

Characteristics.—1. Bacteria are uni-cellular, non-nucleated, usually colorless plants, exceedingly minute.

2. *Composition.* They consist of a substance called myco-protein and probably have a wall of cellulose.

3. *Form.* In form they may be either (1) *rod-shaped* (*bacilli*), or (2) *round* (*micro-cocci*).

Bacilli may be straight, curved or spiral.

4. *Motion.* The round are motionless (except for Brownian movement), the *bacilli* are often motile.

5. *Multiplication* may occur in two ways, (1) by *division*, (2) by *spore* formation.

(1) In *division* the cells may divide and separate from each other, or the new cells may remain united.

In the case of *bacilli* a number may be united end to end in this way, as in *leptothrix*.

In the case of *micrococci* if two are united a *diplococcus* is formed; if a number are united end to end it is called a *streptococcus*, or chain coccus: if a number are united irregularly it is called a *staphylococcus*; if the cells are united in such a way as to form a cube looking like a bale of hay or cotton the collection is called *sarcina*; finally, if a number of cells are held together by a mass of gelatinous intercellular substance, such a mass is called *zooglea*.

The *time* occupied in fission is from ten to thirty minutes.

(2) *Spore* formation may occur in two ways, known, respectively as *endosporous* and *arthrosporous*. In the formation of *endospores* from one to three spots appear in the fungus, and these spots grow and become round or oval in shape; finally the cell wall gives way, and the spores which have developed at the expense of the myco-protein are liberated. In the formation of *arthrospores* one fungus in a chain or cluster becomes larger than the rest and finally

all but this one dies, while it becomes capable of growth to form a new cell.

Most bacteria are probably *monomorphic*, that is, they do not change their form; some are *polymorphic*.

Conditions of Life and Growth.—1. *Food*. The bacteria require nitrogenous food; many develop with especial facility in decomposing animal matter.

Acid fluids are usually unfavorable to their development; *alkaline* are usually favorable.

2. *Water* is essential for the growth and development of all germs, but many of those which are pathogenic may exist in the dry state—for example, the bacillus tuberculosis.

The *endosporous* germs resist drying better than others.

3. *Oxygen* is essential to the life of some germs and prejudicial to others.

4. *Temperature* is of great importance in connection with pathogenic germs; most of them grow and thrive best at a temperature between 98° and 106° Fahr. Many of the *fungi* are killed by freezing, and *boiling* is still more effectual; but the *spores* are very resistant to both heat and cold; they may be frozen up for three months and still live, and prolonged boiling is necessary to destroy them.

5. A state of *rest* is favorable for the development of nearly all the pathogenic fungi.

Distribution of Bacteria in Nature.—*Spontaneous generation* is impossible.

1. *Earth*. Germs are found in great abundance in the surface soil; they disappear, however, about three feet below the surface (Koch).

2. *The bodies of animals* always have germs adhering to them, and disease (scarlet fever, measles, &c.) may be spread in this way.

3. *Clothing* is always contaminated with germs.

4. *Air*. Germs are invariably present in the atmosphere, except in mid-ocean or in deserts. The number of germs in the atmosphere is in direct proportion to population.

5. *Water*. Artesian well water usually contains no germs; water from all other sources does; the water of shallow or "surface" wells is usually rich in germs and dangerous in proportion.

The human body. Germs occur on the *surface* of the body, in the *bronchi* and in the intestinal canal of healthy persons. A few may pass into the tissues, but *in health* they are readily destroyed by the tissues themselves or the leucocytes.

Non-pathogenic germs, often called *saprophytes*, cannot invade and multiply in the living tissues; they can only feed upon dead substances; they are constantly present in the intestines.

Pathogenic germs may invade and multiply in the living tissues. Some seem to be able to act upon perfectly healthy tissues; others

can only act when the vitality of the tissues is already impaired ; for example, diphtheria attacks an inflamed spot more readily than a sound one.

Conditions Influencing the Action of Pathogenic Germs.

These have already been considered under Inflammation (q. v.) and will only be named here—1. *Predisposition*, which may be general or local.

2. *Arrest* of the organisms.
3. The *number* of organisms.
4. The *species* of organisms.
5. The *virulence* of the organisms.
6. *Concurrent growth* of different species.
7. *Local* and *seasonal conditions*.

Effects Produced by Fungi.—1. *General poisoning* by the products of germ action (leucomaines or ptomaines).

2. *Local irritation or inflammation* may result from the direct action of the germs or from the action of the alkaloids formed by them.

3. *Embolism* may occur and lead to secondary troubles, such as pyæmia and multiple abscesses.

Avenues of Introduction of Pathogenic Bacteria.—1. The *respiratory passages*, the germs being taken in with the inspired air.

2. The *digestive canal* with food, water, milk, &c.

3. The *genital passages*, as in cases of gonorrhœal salpingitis or gonorrhœal cystitis.

4. The *skin*, as in ecthyma and impetigo.

5. *Inoculation*, as in vaccination and syphilis.

Modes in Which Pathogenic Bacteria Spread.—1. By *continuity* and *contiguity* of tissue, as in the case of the bacilli of tuberculosis, and the gonococcus.

2. By the *lymphatics*, as in case of syphilis, diphtheria, septicæmia, &c.

3. By the blood vessels, as in cases of septicæmia, &c.

CHAPTER II.

THE ACUTE INFECTIOUS DISEASES.

Definition.—A class of diseases which run a rapid or acute course, each of which is due to an infective agent capable of indefinite multiplication.

Members of the Class.—Typhoid fever, typhus fever, small-pox, scarlet fever, diphtheria, dengue, &c., &c.

Characteristics Common to the Different Members of the Class with respect to—1. *Morbid anatomy*.

- (1) Splenic enlargement;
- (2) albuminoid degeneration of kidneys, liver, &c.;
- (3) liability to inflammation of serous membranes.

2. *Causes*—(1) germ capable of indefinite multiplication;
- (2) infectiveness.

3. *Clinical history*—(1) incubation;
- (2) self-limitation;
- (3) immunity conferred by one attack against subsequent attacks of the same disease (as a rule).

Classification.—1. *Contagious*, in which the germ is conveyed directly or indirectly from one human body to another.

2. *Miasmatic*, in which the germ has no connection with any previous human body.

3. *Miasmatic contagions* (the existence of which is doubtful), in which the germ has to undergo development after passing out of one body before it can produce the disease in another.

TYPHOID FEVER.

Definition.—An acute infectious disease characterized anatomically by inflammation and ulceration of Peyer's patches.

Synonyms.—Enteric fever; abdominal typhus.

Prevalence.—Universal.

Causes.—1. *Germ*, bacillus; in length one third the diameter of red blood corpuscles, width one third its length, motile, contains spores, stains with Bismarck brown, does not liquefy gelatine in culture experiments; the germ has rounded ends and may be

straight or curved. It is found especially in fœcal discharges and in albuminous urine of typhoid patients. Resists cold and heat.

2. *Contagious* if germs get into the body by

- (1) intestinal canal;
- (2) respiratory organs.

3. *Media of Contagion.*

- (1) Drinking water;
- (2) milk and, possibly, meat;
- (3) air, if fœces are allowed to *dry* without being disinfected.

4. *Favorable conditions* for life and development of the germ outside of the body are filth and moisture, but germ may live in the dry state.

The germ may live in ice for months, but requires warmth for its development.

5. *Season of the year.* The disease is most common in the summer or autumn, because the temperature is favorable for development then, and the decomposition of vegetable and animal matter also furnishes favorable conditions.

6. *Age.* Most common between the ages of fifteen and thirty; probably, because persons of this age are more apt to meet with the germ in travel, &c.

7. *Period of incubation*—usually about three weeks, but may be much less.

Morbid Anatomy.—1. *The spleen is enlarged* and softened, because the germs or leucomaines are conveyed to it in the blood and causes congestion and multiplication of cells.

2. *The liver and kidneys* and heart and muscular tissue undergo albuminoid degeneration in consequence of the irritation set up by the leucomaines in the blood; the liver is especially involved, because the leucomaines are carried directly there by the portal circulation, and the kidneys are especially affected, because the leucomaines are eliminated by them.

3. *The larynx, bronchi and lungs* are often inflamed because toxic matters are inhaled from the mouth.

4. *The brain and nervous system* generally show few morbid appearances. *Meningitis* is occasionally present, because of its intimate connection with the lymphatic system, and the germs are diffused through the lymphatics.

5. *The intestines.* The germ usually enters through the mouth; the stomach is but little affected because its acid juice interferes with the development of the germs. The upper part of the small intestine is but little involved, because the bile interferes with the development of germs. The lower part of the small intestine is affected, because the contents of the bowel are alkaline there, the bile has been reabsorbed, and the fœcal matters stagnate there in consequence of the obstruction at the ilio-cæcal valve. Peyer's

patches are especially involved, because the germs are arrested in them.

These patches become red and swollen from the increased amount of blood in the vessels, the exudation of serum and the accumulation of leucocytes. This occurs during the *first* week.

In the *second* week the cells in the patch die from the direct action of the germs and leucomaines and from coagulative necrosis, and a slough is formed which is discharged during the *third* week, leaving an ulcer which usually heals without narrowing of the intestine, but which may eat into a vessel and cause hemorrhage or perforate into the peritoneal cavity.

6. The *mesenteric* glands are frequently inflamed and enlarged, because the germs pass to them from Peyer's patches, are arrested in them and set up inflammation. They may undergo resolution, caseation and calcification, or may soften and burst into the peritoneal cavity.

Symptoms.—A. *Prodromic* or *Premonitory*—not characteristic. Anorexia, headache, chilliness, weakness, and pain in the back and limbs. Bleeding from the nose is quite frequent and suggestive along with the other symptoms.

B. Symptoms of *developed attack*—

1. *Temperature*; gradual rise of 1° each day during the *first* week, the evening temperature being 1° higher than the morning temperature of the same day. In the *second* and *third* weeks a morning temperature of about 103° or 104° and an evening temperature of 104° or 105° . During the *fourth* week a *gradual* fall of temperature, the morning temperature being much lower than the evening temperatures of the same day. A sudden termination of the high temperature is *very rare*, and a sudden fall generally indicates hemorrhage from the bowel or peritonitis. Occasionally, especially in children, the temperature rises suddenly.

2. *Circulatory symptoms*. The pulse is usually from 90 to 100 during the first week. Afterwards it becomes rapid and weak, and sometimes irregular, in consequence of albuminoid degeneration of the heart and muscular coat of the arteries, and, probably, also, in consequence of action of the leucomaines on the intrinsic ganglia of the heart or the vagus nerve.

3. *The skin* is usually hot and dry, but may be wet with sweat when the temperature is high. The amount of heat formed is always above the normal, in consequence of the action of the leucomaines on the heat centres. The sweating sometimes seen is probably due to a paralysis of the inhibitory sweat centres.

An *eruption* of rose-colored spots, few in number, pink in color, about one-eighth inch in diameter, not raised and disappearing temporarily on pressure, is occasionally seen, especially on the chest and abdomen. Each spot lasts for three days and then disappears.

Blood drawn from them shows many typhoid bacilli. Probably a few germs are discharged by the skin.

4. The *urinary* symptoms are, scanty and high colored urine, sometimes containing albumin. It is scanty because the blood pressure is lowered; it is high-colored because the bacilli destroy red corpuscles and consequently more pigment is discharged by the kidneys. The occasional presence of albumin is owing to a slight nephritis, caused by the elimination of leucomaines by the kidneys.

5. The *nervous symptoms* are headache, delirium, which may be mild, or low and muttering, subsultus tendinum, and diminished reflexes. These symptoms are due to poisoning by leucomaines. *Deafness* is not unusual in some cases, and is due to the action of the leucomaines on the nerve centres chiefly, but in part also to inflammation of the eustachian tube.

6. The *digestive* symptoms are anorexia, sometimes nausea and vomiting, diarrhœa, tympanitis, gurgling in right iliac fossa, and sometimes hemorrhage. Occasionally sudden and violent pain. The anorexia and nausea are probably due to the action of leucomaines on the nerve centres; the diarrhœa to the irritation of the bowels by the germs and a serous exudate in consequence; the tympanitis to the fermentation of the contents of the intestine and the loss of tone about the muscular coat of the bowels which permits them to become distended with gas. Gurgling is probably due to the presence of fluid and gas in the cœcum or adjacent parts of the intestine. Hemorrhage is due to the opening of a vessel by ulceration. Sudden and violent pain followed by collapse indicates perforation of the bowel.

The *tongue* is at first coated white and is red at tip and edges; later, brown and dry and cracked. The teeth are covered with sordes from drying of bloody mucus.

7. The *general* symptoms are emaciation and prostration, due to defective nutrition and excessive oxidation and waste.

Symptoms in Atypical Cases.—The *fever* may rise suddenly and such cases usually run a rapid and favorable course.

The temperature may be of moderate intensity, about 101° or 102° throughout the attack. Such cases are tedious but usually end in recovery. In such cases, as a rule, there is constipation and but little delirium.

Constipation is often present even in severe cases. The eruption is very often absent.

Diagnosis.—Often difficult in early stages and even throughout the attack in mild cases.

Distinguished from (1) *acute tuberculosis* by the extensive crepitation in the chest in the latter, (2) *typhoid pneumonia* by the fact that in the latter the pneumonia *precedes* the typhoid symptoms,

(3) *gastro-enteritis* by the different temperature curve in the two diseases.

Prognosis.—Dependent on—1. Type.

2. Duration.

3. Temperature.

4. Complications.

5. Age. Less fatal in children than adults.

Complications.—1. *Pneumonia* from inhalation of morbid matters from the mouth.

2. *Bed sores* from pressure and action of germs.

3. *Parotitis* from entrance of germs into parotid gland through its duct.

Duration.—From three to five weeks—sometimes abortive when it lasts only ten days or two weeks.

Relapses occasionally occur, probably from reinfection.

Sequelæ.—1. *Nervous*, sometimes intellectual weakness of temporary character; occasionally permanent.

2. *Muscular.*—Atrophy of certain muscles occasionally occurs.

Causes of Death.—1. *Toxæmia*, from large amount of ptomaines formed.

2. *Exhaustion*, from fever, diarrhœa or long duration after the attack.

3. *Hemorrhage*, from ulceration of a vessel.

4. *Peritonitis*, from passage of germs into abdominal cavity with or without preforation.

Treatment.—A. *Prophylactic*, attention to be paid to water supply, sewer pipes, &c., to prevent contamination with germs.

Fæcal discharges to be disinfected by solution of bichloride of mercury, 1 to 500, or chloride of lime, 1 to 100.

Bed clothes to be disinfected by soaking in bichloride solution, 1 to 1000, and subsequent boiling.

B. *General management.*—1. Room should be large, to furnish fresh air.

2. Patient should be kept in bed strictly, because movement increases exhaustion.

3. Diet should be *liquid* and nutritious, such as half a glass of milk or *fresh* buttermilk every four hours.

C. *Medicinal treatment* directed—

1. To *reduce temperature*, if over 103° , by cold or cool baths, unless hemorrhage has occurred, when they are dangerous, antipyrine, antifebrine, phenacetine, and quinine.

2. To *sustain strength*, by stimulants and nourishment.

Stimulants not always necessary. If they cause quickening of

the pulse, the quantity should be diminished, or they should be stopped altogether.

D. *Specific* treatment is intended to kill the germ. It is of doubtful value. The germicides which have been used are—

1. Calomel.
2. Iodine.
3. Carbolic acid.
4. Corrosive sublimate.
5. Naphthaline, &c.

In many cases of a mild character no medicinal treatment is necessary; but the importance of *care in diet* cannot be exaggerated. Perforation of the bowel may occur from imprudence in the mildest cases. Absolute rest in bed is of almost as great importance.

Treatment of Special Symptoms and Complications.—1. *Diarrhœa.*—Opium, bismuth and other astringents to check secretions and naphthalin to check fermentation and probably destroy germs.

2. *Tympanites.*—Turpentine internally and turpentine stupes to stimulate the muscular coat of intestine. Aspiration and the insertion of a tube into the rectum to withdraw the gas.

3. *Hemorrhage.*—Opium to paralyze the muscular coat of the bowels and absolute quiet to allow a clot to form. Ergot to cause contraction of the vessel and turpentine for the same purpose.

4. *Peritonitis.*—Opium to relieve pain and paralyze the bowel so as to prevent escape of fecal matter as far as possible. Laparotomy has not given good results because the patients are already exhausted.

5. *Bronchitis and pneumonia* to be prevented by cleansing the mouth frequently; treated chiefly by stimulants and dry cupping. Stimulants help the heart to force the blood through the diminished respiratory space.

6. *Bed sores* to be prevented by frequent changing of position, air bed and sponging with alcohol and corrosive sublimate; treated antiseptically by bichloride solution 1 to 2000 or 4000 and iodoform ointment.

7. *Constipation* to be relieved by enemata because of the danger of profuse diarrhœa or perforation if purgatives are used.

8. *Delirium*, if mild, to be treated by cold to the head, phenacetine or opiates; if low and muttering, by stimulants and opium.

Sleeplessness to be treated by opium, paraldehyde, and if the pulse is full and strong, by chloral.

Management and Diet During Convalescence.—Patient should remain in bed and take liquid food only for at least a week after the evening temperature is normal because of the danger of preforation of the bowel.

TYPHUS FEVER.

Definition.—An acute infectious disease characterized by a rapid rise of temperature which lasts about two weeks and then suddenly declines, violent headache followed by delirium and a macular eruption which appears on the fifth or sixth day and persists until the end of the attack.

Synonyms.—Jail fever, ship fever, spotted fever, &c., &c.

Causes.—1. A germ probably, but it has not yet been discovered. The germ seems to require filth for its existence outside of the body.

2. The *favorable conditons* for the communication of the disease are over-crowding and bad hygienic surroundings and also the concentration of the poison or germs.

Over-crowding and bad hygiene act by diminishing the resisting power of the individual and probably also by furnishing a suitable soil for the life of the germ. When the poison is concentrated or the germs numerous it is probable that the phagocytes cannot dispose of them.

3. The *avenue of introduction* is the respiratory mucous membrane.

4. The *medium of contagion* is the air—the exhalation from the lungs and probably from the skin.

5. One attack confers *immunity* as a rule from subsequent attacks.

6. The *period of incubation* is usually about two weeks.

Morbid Anatomy.—1. The *blood* is dark in color and deficient in fibrin factors, probably from the action of the bacteria or leucomaines.

2. The *heart* and *general muscular tissue*, *liver* and *kidneys* show albuminoid degeneration and the spleen is enlarged as in the other acute infectious diseases and for the same reason.

3. The *lungs* are frequently inflamed from the inhalation of decomposing substances from the mouth or from the circulation through them of leucomaines.

4. The blood vessels of the *brain* are engorged and often there is a serous exudate.

5. There are no characteristic changes in the *intestinal canal*.

Complications.—1. *Bronchitis*, *pneumonia* and sometimes *pulmonary edema* and also *pleurisy*. The occurrence of the former has been already explained. The latter is due to the connection of the pleural cavity with the lymphatic system.

2. *Meningitis* occasionally occurs for the same reason that pleurisy does.

3. *Glandular enlargements* are due to the passage of bacteria and leucomaines through the lymphatic vessels and their temporary arrest in the glands with consequent inflammation.

4. *Thrombosis* of the veins of the lower extremity is due to pressure on the veins by enlarged glands and defective nutrition of the vascular walls.

Symptoms.—1. *Mode of onset* is sudden.

2. *Nervous symptoms*, chills, headache, backache, delirium, stupor and coma vigil due to poisoning by leucomaines.

3. The *temperature* rises suddenly to 103° – 105° , or even 106° , and falls in favorable cases on 11th to 14th day. The fever is due to the action of leucomaines on the heat centres in the brain.

4. *Digestive symptoms*. Sometimes there is nausea, probably central in origin and due to the action of leucomaines on the brain.

Constipation is due to albuminoid degeneration and weakening of the muscular coat of the bowels.

5. *Circulatory symptoms*. The pulse ranges from 100 to 130 usually; the increased rapidity is due to increased temperature and probably also to direct action of leucomaines; it soon becomes soft and compressible from changes in the heart and vessels.

6. *Cutaneous symptoms*. An eruption appears on the 5th or 6th day on chest and abdomen, macular in character, dark in color and each spot lasts throughout the disease.

7. *Urinary symptoms*. Albuminuria, diminution in quantity and complete suppression may occur from the changes in the kidneys. There may be retention of urine from paralysis or benumbing of nervous sensibilities.

Diagnosis from—1. *Meningitis*, made by the presence of a rash in typhus fever and the greater prostration and more rapid pulse.

2. *Typhoid fever*, by the absence of diarrhœa, the different course of the temperature, and the difference in the eruption.

3. From *relapsing fever*, by the eruption of typhus, and the absence of relapses.

Prognosis.—The *prognosis* is based on *age*, the mortality in children being very low; *previous health* and *habits as to temperance*. It is exceedingly fatal in the intemperate, because the kidneys are often diseased in such persons, the blood-vessels are degenerated and the resisting power of the tissues generally is impaired.

Causes of Death.—1. *Toxæmia*, from the amount of poison (leucomaines) formed.

2. *Syncope*, from the action of the leucomaines on the muscular tissue of the heart and probably on the intrinsic ganglia.

3. *Complications*, such as pneumonia and meningitis.

Duration.—Usually about *fourteen days*, when the disease terminates by *crisis*.

Treatment.—A. *Preventive.*—1. *Quarantine*.

2. *Disinfection* of the room or house with sulphurous acid or chlorine.

3. *Ventilation*, which seems to scatter the germs so that only a few are taken into the body, and they are probably destroyed by the phagocytes.

B. *Remedial*.—The indications are—

1. To *neutralize the poison*, which can best be done by *fresh air*.
2. To *reduce the temperature* by cool sponging, phenacetine, &c.
3. To *sustain strength* by food and stimulants, if the latter do not increase headache and quicken the pulse; if they do, they should be discontinued.

RELAPSING FEVER.

Definition.—An acute, infectious and contagious disease, characterized by high fever, great prostration, rapid *apparent* recovery, and after a week or ten days a relapse.

Synonyms.—Famine fever—recurrent typhus.

Causes.—The essential cause is the *bacterium* known as the spirillum obermeieri, which is found in the blood during the attack, and is motile. It is propagated by contagion; enters by the respiratory mucous membrane; one attack is protective. Incubation from one to seven days.

Morbid Anatomy.—Not characteristic. The changes common to the acute infectious diseases are found.

Symptoms.—1. *Nervous*; chill, headache, pain in the back and limbs from poisoning.

2. Rapid rise of *temperature* to 104° to 107° from action of leucamines on the heat centre.

3. *Digestive*.—Nausea and vomiting, constipation.

4. *Circulatory*.—Rapid and feeble pulse from condition of heart and vessels.

5. *Remission and relapse* from causes which are not known.

Diagnosis, based on prostration, character of the fever, absence of eruption of typhus, and relapse.

Prognosis is very good; mortality about 3 per cent.

Treatment.—Indications.—1. To *relieve pain* and *reduce fever*, by phenacetine, antipyrine, &c.

2. To guard against syncope, by absolute quiet.

SCARLET FEVER.

Definition.—An acute, infectious and contagious disease characterized by a rash at first punctate and not raised, which runs a definite course and is followed by desquamation.

Varieties.—1. *Simple*, when it pursues a mild course.
2. *Malignant*, when it pursues a very severe course.

Causes.—1. A *germ* is almost certainly the essential cause, but it has not yet been discovered with certainty.

2. *Media of Contagion*, the atmosphere, clothing, letters, milk animals, &c., the germ being contained in the mucous secretions, blood and epidermic scales.

3. *Avenues of Introduction*, the lungs, the stomach and the skin (by inoculation.)

4. *Immunity* is conferred by one attack, as a rule, against subsequent attacks.

5. *Age*—It is rare in children under six months old and in adults.

6. The *period of incubation* is from a few hours to ten days.

Morbid Anatomy.—1. The changes common to the acute infectious diseases are found in scarlet fever.

2. The *skin* exhibits an eruption, at first punctate in character afterwards becoming diffused.

3. The *tonsils and throat* are inflamed and swollen, and the tissues of the neck may be infiltrated with sero-fibrinous exudate or abscess may form in consequence of the passing of the germs from the throat into the lymphatics and tissues.

3. The *glands* around the throat may be inflamed and swollen from the same cause.

4. The *kidneys* are often inflamed from the elimination of germs and leucomaines through them.

Symptoms.—A. *Prodromic*. Sore throat is the most conspicuous, due probably to the arrest of the germs there and the presence of favorable conditions for their development and growth.

B. *Developed*. 1. *Nervous*. Those characteristic of the acute infectious diseases already mentioned.

2. The *temperature* rises rapidly to 103 or 105; the skin feels singularly hot.

3. *Circulatory*. The pulse is very rapid and becomes weak.

4. *Digestion*. Nausea and, sometimes, vomiting; strawberry tongue, from enlargement of the papillæ.

5. *Eruption*, which appears on the second day, first on the neck and chest, is scarlet in color, punctate at first, and then becomes uniform by coalescence, begins to fade on the fourth day, and desquamation begins on the 6th or 8th day and lasts two weeks.

Malignancy may take the form of toxæmia, a very high temperature, a dark eruption or great involvement of the throat.

Mild cases may occur with and without eruption. Peeling may occur when there has been no eruption and such cases may cause others of malignant character.

Complications.—1. *Nervous convulsions or mild delirium or stupor* may occur from (1) poisoning by leucomaines, (2) interference with the circulation by swelling of the tissues of the neck, (3) uræmia.

2. *Otitis media*.—Inflammation of the middle ear is common from the germs passing up through the eustachian tube.

Deafness often ensues and young children may be *dumb* also in consequence.

3. *Diphtheria* is an occasional complication.

Sequelæ.—1. *Nephritis*, leading to more or less extensive dropsy, is common and is probably due to the irritation of the kidneys by the leucomaines during their excretions by these organs.

2. *Inflammation of serous membranes*, especially the endocardium, is of comparatively common occurrence and is due to the leucomaines in the blood.

Pleurisy and rheumatism are also occasional sequelæ. Rarely there is suppurative inflammation of the joints.

Diagnosis.—1. From *measles* it is distinguished by the absence of the premonitory coryza and the punctate or non-elevated eruption.

2. From *small-pox* by the difference in the eruption, absence of vesicles or pustules.

3. From *erythema and roseola* by the absence of punctate redness at any stage of the latter affections.

4. From *diphtheria* by the eruption in scarlet fever; if there is no eruption a diagnosis is extremely difficult.

Prognosis.—The *prognosis* is always uncertain; the mortality ranges from five to twenty per cent. It is greatest in children between one and five years of age. It is further dependent on (1) the character of the epidemic, (2) the severity of the throat lesions, (3) the height of the temperature.

Treatment.—A. *Prophylactic.*—1. *Isolation* is by far the best.

2. *Disinfection* of the room and clothing by chlorine gas and solution of bichloride of mercury is essential.

3. *Boracic acid*, possibly, may prevent the disease in children who are exposed to it (Lewis Smith).

4. *Oiling the surface* of the body prevents the scales from being disseminated as readily by the atmosphere, and hence acts as a prophylactic.

5. *Ventilation* is always advisable.

6. *Belladonna* is useless.

B. *Medicinal.*—1. To *neutralize the poison* of the disease as far as possible by *ventilation*; *boracic acid* may lessen the virulence of the poison also. Bichloride of mercury and muriated tincture of iron are used for the same purpose.

2. To *reduce fever* by cold sponging, cold baths if the temperature is over 105, (there is more danger of nephritis probably when cold baths are used), phenacetine, acetanilide, &c.

3. To *promote the action of the skin* by sponging with tepid water.

4. To *relieve itching and burning* by sponging with tepid water, or saline solution, or weak alcoholic solution.

5. To *relieve throat complications* by the use of sprays of listerine or carbolic acid, &c., or by the administration of bichloride of mercury and muriated tincture of iron in glycerine.

6. To *prevent nephritis*, by promoting the action of the skin and by the avoidance of chilling of the surface.

7. To *sustain strength* by suitable food and stimulants.

DIPHTHERIA.

Definition.—An acute, infectious and highly contagious disease characterized anatomically by the formation of a false membrane on some of the mucous surfaces, especially that of the pharynx.

Causes.—1. A *germ*, probably a streptococcus, which finds *favorable conditions* for its development outside of the body in filth, especially sewage.

The favorable conditions for the occurrence of the disease in an individual are depression of the general health and some inflammation, acute or chronic, of the pharyngeal mucous membrane.

The *avenues of introduction* of the germ are (1) the respiratory mucous membrane;

(2) the mucous membrane of the mouth and throat;

(3) probably also an abraded surface.

The *media of contagion* are: (1) the atmosphere; very often sewer gas contains the germs of diphtheria;

(2) the mucous secretions and probably the blood;

(3) clothing and other substances, and also animals; it is probable that certain animals, especially birds, are liable to this disease and may communicate it to man.

2. *Age.* The disease is more common between the ages of six months and two years, but may occur at any period of life.

3. One attack of diphtheria does not confer immunity from subsequent attacks.

4. The period of *incubation* is from one to eight days, rarely longer.

Morbid Anatomy.—1. The *false membrane*—

(1) Its *situation*. It may be found on *any* mucous surface, or any abrasion or wound;

(2) *mode of formation and structure*. It consists of epithelial cells and leucocytes, which are granular and degenerated, entangled in a network of fibrin. A *croupous* membrane is on the *surface* of the mucous membrane; a diphtheritic membrane is not only on the

surface, but extends *into the substance* of the mucous membrane. The membrane is formed by coagulation necrosis and by the exudation of fibrin;

(3) *color* and *thickness*. The color is an ashy gray, and the membrane is often a fourth of an inch in thickness;

(4) *bacteria* of various kinds are formed in the membrane. The coccus forms predominate;

(5) the *mode of removal* of a diphtheritic membrane is by absorption, suppuration or gangrene.

The membrane is so closely adherent to the tissues that it cannot be removed without tearing it off, and after being removed in this way it soon returns.

It may undergo *absorption* in mild cases, softening first occurring, and the softened material is taken up by the lymphatics and blood vessels.

Suppuration often occurs, the membrane being thus loosened from the tissues beneath it.

Gangrene may occur in bad cases from the pressure upon the blood vessels, and probably also from thrombosis of the vessels in the inflamed tissues.

2 The *heart, kidneys, muscles* and *liver* show *albuminoid* degeneration which may pass into *fatty* degeneration.

3. The *spleen* is enlarged and the lymphatic glands in the neighborhood of the inflamed surface are inflamed and swollen. The tissues around the glands are also inflamed and swollen; the inflammation is due to the action of the germs and the leucomaines formed by them, and the swelling is due to the exudate.

4. The *brain* and *nervous system* are often involved. A neuritis is frequent, which is probably due to the action of leucomaines, but the morbid changes in the nervous system are not clearly understood.

Symptoms.—The *symptoms* present no typical course, but vary greatly in character and intensity in different cases.

A. The *local* symptoms: 1. When the *pharynx* is involved, are redness of the throat, which is very dark in color and often circumscribed; later on, an ashy membrane forms on the tonsils or pharynx; there is some pain but in many cases it is not severe. The glands of the neck are swollen and the tissues infiltrated with inflammatory exudate, because the germs have passed from the mucous membrane of the throat to the glands, been arrested there and set up inflammation. They have gone into the surrounding tissues through the lymphatic vessels.

2. When the *nose* is involved, it is stopped up by the swollen mucous membrane and the false membrane; there is a serous and often slightly bloody discharge which excoriates the upper lip on which a false membrane forms. The tissues underneath the angle of the jaw are greatly swollen.

3. In *laryngeal diphtheria* there are difficulty of breathing, a hoarse muffled cough, blueness of the skin, sometimes convulsions followed by coma and death. All these symptoms are due to the formation of false membrane on the vocal cords which prevents the entrance of air and the proper oxygenation of the blood.

B. The *constitutional* symptoms vary very much in different cases.

1. The *temperature* has no typical course; it may reach 103° or 104° , or it may not be elevated above 100° . The degree of temperature has no prognostic significance.

2. The *circulatory* symptoms are often striking. As a rule the pulse becomes very feeble and rapid, but in some cases after the membrane has disappeared from the throat the pulse may become very slow, sometimes falling to thirty beats per minute. Such cases rarely recover. The weakness of the pulse is due chiefly to the albuminoid or fatty degeneration of the heart. The change in the number of beats is due in all probability to nervous influence, but it is not known what the changes in the nervous system are.

3. The *digestive* symptoms are anorexia and sometimes nausea and vomiting. These symptoms are probably due to the action of the leucomaines on the nerve centres.

4. The *urinary* symptoms. In many cases there is albuminuria, and sometimes casts are found in the urine.

5. The *general* symptoms are prostration, which is often extreme, and, in many cases, pallor.

Complications.—*Pneumonia* is not uncommon. It is due to the inhalation of noxious matters from the mouth, larynx and bronchi, the diphtheritic process often extending into the bronchial tubes.

Conjunctivitis is apt to occur either by extension through the tear duct or by some of the discharge from the mouth getting into the eye.

Otitis media is a common complication, the germs passing up through the eustachian tube.

Gangrene of the fauces and tissues of the neck occurs in bad cases from pressure on the blood vessels and thrombosis and probably also from the immediate destruction of the tissues by the concentrated leucomaines.

Inflammation of serous membranes may occur as in the other acute infectious diseases. The manner in which they are produced has already been explained.

Sequelæ.—1. *Paralysis* is the most common and striking sequel. The parts usually involved are the muscles of the *palate and of the eyes*. Sometimes the muscles of the extremities and body are affected. The tendon reflexes are lost. The paralysis is probably due to a degenerative neuritis, but it is not clearly understood. Unless the heart or the muscles of respiration are involved, diphtheritic paralysis nearly always terminates in recovery. 7

2. *Parenchymatous nephritis* sometimes occurs in consequence of the elimination of the leucomaines by the kidneys.

3. *Deafness* may be caused by the closure of the eustachian tube from cicatricial contraction, or it may be due to the loss of the drum-head and the small bones of the ear.

Diagnosis.—1. *Follicular pharyngitis* causes sore-throat and the tonsils often present upon their surface yellow or white spots or a yellow coating; but this coating is readily removed by a brush while the membrane of diphtheria is not.

2. *Scarlet fever* also causes sore throat, but nearly always there is the characteristic eruption and there is, as a rule, no false membrane.

Prognosis.—The *prognosis* of diphtheria is *always uncertain*, even from hour to hour. It is dependent on

1. *Age*, the mortality being greatest between the ages of six months and ten years.

2. *Character of the epidemic.*

3. *Complications.*

4. *Involvement of the larynx.* Recovery in cases of laryngeal diphtheria, unless the patient is subjected to tracheotomy or intubation, is exceedingly rare.

5. *Location and atmospheric conditions.* The mortality is very great in high and cold situations.

Causes of Death.—1. *Toxiemia*, from the amount of leucomaines absorbed.

2. *Asphyxia*, from obstruction of the larynx, or from paralysis of the respiratory muscles.

3. *Exhaustion*, from defective nutrition and heart failure.

4. *Paralysis of the heart.*

5. *Complications.*

Treatment.—A. *Prophylactic.* 1. *Isolation*, to prevent the communication by direct contagion. The germs are not carried far by the atmosphere.

2. *Disinfection* of sputa and discharges by bichloride of mercury, &c.

3. *Disinfection of clothing* by bichloride of mercury solution or chloride of lime and by boiling.

4. *Disinfection of the room* by washing the walls with bichloride solution and by fumigation with chlorine or sulphurous acid.

B. *Remedial*—

1. *Constitutional*—(1) to *sustain strength* by alcohol and nutrient and by muriated tincture of iron, &c.;

(2) to *destroy* or *weaken* the germs (?) by the use of bichloride of mercury in large doses, and benzoate of soda, &c., &c.

2. *Local.*—(1) To *reduce inflammation* as far as possible by hot

application or poultices applied to the neck. No counter-irritants should be used because the false membrane forms readily on an abraded surface.

(2) To *dissolve* or *loosen* the *false membrane*, by inhalation of steam, turpentine, carbolic acid, or by sprays of carbolic acid, lime water, lactic acid, trypsin or papayotin. The three latter agents will help to dissolve the membrane.

(3) To *disinfect* the *throat* by bichloride of mercury, carbolic acid, listerine, &c.

Nasal diphtheria is to be treated by frequent and thorough syringing of the nose with antiseptic solutions.

Laryngeal diphtheria should be treated by—

(1) intubation, or

(2) tracheotomy.

The percentage of recoveries is about the same after each operation, namely, twenty-eight.

DYSENTERY.

Definition.—An acute infectious disease characterized by inflammation of the large bowel and frequent actions of mucus and blood.

Forms.—The disease may be *epidemic* or *sporadic* and it may be *acute* or *chronic*.

A disease precisely similar in symptoms and morbid anatomy to infectious dysentery, and which is commonly called dysentery, may be caused by constipation and other things without the direct action of germs so far as our present knowledge is concerned.

Causes.—1. A *germ* in all probability is the essential cause of acute epidemic dysentery, but it has not yet been discovered.

2. The *favorable conditions* for the development of the infective agent are warmth, moisture and filth, hence the disease is most common in hot climates or hot seasons, in damp localities and where animal or vegetable matters are undergoing decomposition.

3. The *avenue of introduction* is the alimentary canal.

4. The *medium of conveyance* is usually water. The germ is discharged from the body with the feces and if these germs gain access to the drinking water the disease will occur in those using such water. The germ, like that of typhoid fever, is apparently destroyed, or at any rate rendered inert, by drying.

Morbid Anatomy.—1. The *large bowel* is the part involved. The change there is inflammatory in character. Redness, swelling and exudation occur. The cause of these has frequently been ex-

plained. The exudation is usually fibrinous in character and leads to sloughing, and the solitary and agminated glands, when the germs are arrested, become inflamed, swollen and ulcerated. Later the ulcers heal and cicatricial contraction results.

2. *Complicating conditions* of occasional occurrence are *abscess of the liver* from the lodgment in that organ of the septic emboli, brought by the portal vessels from the large bowel, and inflammation of serous membranes, which are occasionally seen in all the acute infectious diseases.

Symptoms.—1. *Nervous.* A chill often occurs at the commencement, pain and tenderness over the region of the large bowel in consequence of the inflammation there.

2. *Digestive.* The appetite is lost, probably from the action of the toxic matters generated by the germs. The tongue is heavily coated usually, and, if the case assumes a typhoid character, it becomes brown and dry, and sordes collect on the teeth. Often there is troublesome nausea and vomiting. The stools are at first feculent, but soon consist of blood and mucus. The quantity discharged each time is small but the actions are frequent. The blood comes from the bursting of the engorged vessels and ulceration, the mucus from the excessive secretion in the large bowel. The frequent desire to evacuate the bowel and the straining or tenesmus at the time is due to increased reflex excitability in consequence of inflammation.

3. The *temperature* is elevated, being usually between 103° and 104° .

4. The *circulatory* symptoms are, rapidity of the pulse, which, however, is not very marked in the early stages; but later there is great frequency and feebleness from the changes in the heart muscle, which are common to all infectious diseases.

5. The *urinary* and *vesical* symptoms are often very marked; the urine is scant and dark colored, and there is frequent desire to pass water with spasm at the neck of the bladder, which is reflex in character, and is due to the inflammation of the rectum.

In malarial sections, or in persons who have suffered with malaria, the disease assumes an intermittent or remittent character.

Diagnosis.—1. From *acute intestinal* catarrh dysentery is distinguished by the absence of mucus and blood and of tenesmus in the former disease.

2. From *diarrhœa* complicated with hemorrhoids it is distinguished by the fecal character of the discharges in diarrhœa and the absence of tenesmus.

Course.—Dysentery usually pursues a typical course and the duration of the attack is about ten days.

Prognosis.—The prognosis depends on—1. The type of the disease.

2. The *previous health* of the patient.

3. The *complications*.

As a rule the disease ends in recovery, but may assume a chronic form.

Treatment.—*Prophylactic.* The *prophylactic* treatment is precisely the same as that of typhoid fever, and consists in purification of the water if contaminated, disinfection of the stools and clothing and the avoidance of cold and dampness and of improper food, both of which are liable to cause congestion of the bowels and thus render them more vulnerable.

Remedial treatment consists in—1. *Sustaining strength* by nourishing food and stimulants.

2. *Removal of offending matters*, such as scybala from the bowels by saline aperients, castor oil or enemata.

3. *Relieving pain* by hot applications to the abdomen, opiates and bismuth.

4. *Checking the discharges* by opium and bismuth and astringents.

5. *Disinfecting the bowel* by means of bichloride of mercury or naphthalin or by enemata or by chloride of mercury or by irrigation of the bowel with simple water.

6. *Specific treatment* with ipecac which is sometimes given in large doses, but seems equally beneficial in smaller quantities.

In chronic dysentery injections of silver nitrate are very useful.

EPIDEMIC CHOLERA.

Definition.—An acute infectious disease, characterized by profuse watery action from the bowels, great prostration and cramps in the limbs.

Distribution.—Its home is in India, and it is a disease of hot countries chiefly.

Synonyms.—Asiatic cholera, or simply cholera.

Causes.—1. A *germ* which has been completely identified is the essential cause. This germ is a curved bacillus, and is hence called the comma bacillus; it is about half the length of the bacillus tuberculosis, and is motile. The germ is found in the discharges from the bowel and in the contents of the bowel after death, but not in the walls of the intestines.

2. The *favorable conditions* for development are *moisture*, which is essential to the life of the germ, a certain degree of *warmth* and organic matter. Sewage is a very favorable soil for the development.

3. The *avenue of introduction* is in all cases, perhaps, the alimentary canal.

4. The *medium of conveyance* is in all, or nearly all, cases, perhaps, drinking water; because the germ is contained in the fecal discharges, and the water is liable to be contaminated by them; but persons handling the moist clothes of cholera patients may get the germs on their hands and thus get them in the mouth, and contract the disease in this way.

5. The *period of incubation* is from a few hours to a few days.

Morbid Anatomy.—1. The *general appearance* of the body is very striking; it is very white and shrivelled.

2. The *small intestine* is softened somewhat, its epithelium is in part desquamated, and it contains a rice-water fluid with flakes of epithelium in it.

3. The *kidneys* are small and dark, and the bladder is empty because of the loss of fluid.

4. The *lungs* and *bronchi* are also much harder and dryer than usual for the same reason.

5. The *heart* and *spleen* undergo a similar change, the latter organ being smaller than normal, on account of the loss of water from the body.

Symptoms.—*Prodromic*, or 1st stage. The prodromic symptoms are slight, and consist of a painless diarrhœa. This is frequently called the first stage.

In the *2nd stage* the symptoms are well marked.

1. *Digestive.*—There are profuse water actions at first containing some feces and bile, but later resembling rice water in appearance and being devoid of odor. They contain a very large amount of albumin.

Vomiting also occurs, large quantities of fluid being ejected.

The profuse watery discharges are probably due to a paralysis of the nerves of the bowel from the action of the toxic substance generated by the comma bacilli.

2. The *temperature* is elevated, but is not usually very high, and the extremities feel cool in consequence of the extreme feebleness of the heart and the loss of the water from the blood.

3. The *pulse* is very rapid and feeble in consequence of defective nutrition of the heart muscle and the changes in that organ, and probably also in consequence of the direct action on the heart of the toxic substance.

4. The *urine* is scant and high colored, because the blood pressure is lowered and but little water passes out through the kidneys.

5. *Nervous* and *special sense* symptoms are present. There are violent cramps in the limbs and body, especially in the calves of the legs, and the sufferers may become deaf and blind. The second stage lasts from one to two days.

In the 3rd or *algid stage* all the symptoms are aggravated. The

discharges from the bowels continue, the pulse becomes imperceptible, the extremities are cold and there is suppression of urine. This stage rarely lasts more than six or eight hours and is succeeded by death or the *reactionary stage*.

Reaction is often rapid, and is marked by improvement in all the symptoms.

Cholera typhoid may come on after reaction has occurred. The temperature rises, the bowels become loose again and the actions are often bloody, and these are the usual typhoid symptoms.

Occasionally *cholera nephritis* occurs and causes death by urinæmic poisoning.

Diagnosis—1. *Arsenical poisoning* is distinguished from cholera by the absence of blood and feculent matter in the actions in the latter disease, but there is often a striking similarity between the two.

2. From *gastro-enteritis* cholera is distinguished by the different character of the actions and by the difference in the course of the two diseases.

Prognosis.—The *mortality* varies from twenty to eighty per cent. As a rule the whole course of the disease is rarely more than five or six days, though reaction is occasionally slow.

Treatment.—*Prophylactic* treatment consists in—1. *Quarantine*.

2. *Disinfection* of stools by bichloride solution and of clothing by dry heat.

3. *Drainage and cleanliness*.

4. *Diet*. The utmost prudence should be observed during a cholera epidemic.

Remedial treatment consists in 1. *Controlling the premonitory diarrhœa* by quiet, opium and astringents. Large enemata of tannic acid seems to be especially efficacious and are thought to destroy the comma bacilli.

2. *Relieving symptoms* by stimulants, warmth to the extremities and suitable diet during reaction.

YELLOW FEVER.

Definition.—An acute, infectious disease, conveyed by fomites, occurring usually in hot countries, and characterized by a fever of short duration, usually yellowness of the skin, great prostration and in some cases "black vomit."

Causes.—The *essential cause* is almost certainly a *germ*, but it has not yet been discovered with certainty,

2. The *favorable conditions* for its *development* are warmth, moisture and filth. Overcrowding is also a factor in its production.

Cold destroys the germ, the first frost usually causing an abrupt termination of an epidemic.

3. *Modes of conveyance* of the poison. The germ may be carried by persons sick with the disease in clothing, hair and probably in letters. It is also carried in the atmosphere, but seems to be confined to the lower strata, and is readily stopped by streams, forests or even streets.

4. With respect to *race* it has been found that negroes are less liable to the disease than white persons and have it less severely.

5. The *period of incubation* is from one to five days.

6. One attack is protective, as a rule.

Morbid Anatomy.—1. The *blood* is dark in color and does not coagulate readily.

2. The *heart, kidneys, spleen and liver* undergo degenerative changes as in the other infectious diseases. The *liver* in yellow fever, however, is of a yellow color, and its cells are filled with fat.

3. Infarctions of the *lungs* are of frequent occurrence.

4. The *mucons membrane* of the stomach and bowels is greatly congested.

5. The *skin* in most cases is of a deep yellow color, and the conjunctivæ are similar in appearance.

Symptoms.—1. *Nervous*; chilly sensations, sometimes chills, headache, pain in the back and limbs and tenderness over the stomach are the most important and common nervous symptoms. In severe cases delirium occurs and may be very troublesome.

2. The *temperature* ranges from 102° to 105°, and rises rapidly to this point. There is usually a remission on the fourth or fifth day and in mild cases this is the beginning of convalescence.

3. The *circulatory* symptoms are, quickening of the pulse and weakness of the heart's action. The pulse is, however, slower in yellow fever than in the other acute infectious diseases, and this fact is apparently connected with the jaundiced color of the skin.

4. The *digestive* symptoms are *nausea* and *vomiting*, and usually constipation. The vomited matters are in mild cases very bilious in character, and in some cases contain blood, constituting the dreaded black vomit.

5. The *urinary* symptoms are *scantiness* and in some cases suppression of urine. There is sometimes albuminuria.

6. The *skin* is usually of a yellow color and perspiration is common.

Diagnosis.—The diagnosis is based on—1. The black vomit.

2. The range of temperature.

3. The color of the skin.

4. The suppression of urine.

The Prognosis is always very serious, but differs in different epidemics. The mortality ranges from 10 to 30 per cent.

The Duration is usually about six days, but convalescence is often tedious.

Treatment.—The *prophylactic* treatment consists in—1. *Quarantine*.

2. *Disinfection of clothing, &c.*

The *remedial* treatment.—Diaphoretics have been found useful. They probably act by lessening the work of the kidneys.

Restlessness and nausea should be relieved by morphia and champagne or brandy.

MALARIAL DISEASES.

Definition.—Affections differing very much in character, but all of which are due to a poison generated in warm and damp localities, especially in marshy places.

Causes.—1. A *germ* is the essential cause; probably that described by Laveran; it may occur in several different forms. Sometimes it is found in a red blood corpuscle, when it is amœba like in character, capable of slow movement, and causes destruction of the corpuscle with liberation of pigment; there may be but one germ in a corpuscle, or there may be several. Sometimes these germs contain pigment which is arranged in the form of a rosette. Crescent-shaped germs, with clubbed ends, are found free in the blood, and now and then oval, or round, or pear-shaped parasites, with flagellæ.

2. The *favorable conditions* for the *development* of these germs are warmth and moisture. Marshy ground, which is sometimes covered with water and sometimes exposed to the sun, is an especially favorable soil for their development. The conditions which render a *person* liable to malarial attacks are *debility* from any cause and a *previous attack*.

3. The *avenues of introduction* of the germs are the respiratory mucous membrane and the intestinal canal.

4. The *media of conveyance* are—

(1) *atmospheric air*; the germs are sometimes carried considerable distances by currents of wind, but they are contained in the lower strata of the atmosphere, and hence persons on high land or in the upper story of a house are not so liable to take the disease as those at a lower level;

(2) *waters* from malarial districts may carry the germ for long distances;

(3) *milk* is probably also a medium of conveyance;

(4) possibly certain *articles of diet* may act as media of conveyance.

5. One attack does *not* confer immunity from subsequent ones,

but, on the contrary, increases the liability to them.

6. The *period of incubation* is not fixed ; it probably varies from five to thirty days.

There is sometimes *apparent tolerance* of the malarial poison when persons living in a malarial country do not suffer from malarial affections, but the course of any disease occurring in such persons is usually modified by the malaria.

Latency is said to exist when there is no evidence of malarial poisoning for some time after exposure, but when subsequent developments prove its existence.

Forms of Malarial Diseases.—The following forms are comparatively common :

1. Intermittent fever.
2. Remittent fever.
3. Typho-malarial fever.
4. Chronic malarial toxæmia.
5. Pernicious malarial fever.

INTERMITTENT FEVER.

Definition.—An acute infectious disease due to the presence of the malarial germ and characterized by periodical recurrences of chills, fever and sweating, the person being free from fever in the interval.

Synonyms.—Ague and fever. The shakes, and chills and fever, &c.

Causes.—The causes are those of malarial affections generally.

Types.—1. Quotidian.

2. Tertian.

3. Quartan.

4. Double forms.

5. Dumb ague and masked forms.

6. Those in which there is a tendency to recurrence every seven, or fourteen, or twenty-one days.

Symptoms During a Paroxysm.—1st, or *cold stage*, lasts about one or two hours—

(1) the *skin* is cool and covered with “goose-bumps” ;

(2) the *internal temperature* is increased, while that of the surface and extremities is depressed ;

(3) the *nervous* symptoms are a *chill* and pain in the head, back and limbs, and a feeling of extreme coldness ;

(4) the *digestive* symptoms, nausea and vomiting are common.

The 2nd, or *hot stage*, lasts five or six hours usually—

(1) the *skin* during this stage becomes red and hot ;

(2) the *temperature* is elevated to 104° or 105° or even to 106° or 107° ;

(3) the *pain* in the head and back and limbs is intense.

4. The *circulatory* symptoms are fullness and quickness of the pulse, which often reaches 110° or 120° .

5. The *digestive* symptoms are nausea and vomiting.

In the *third, or sweating* stage, there is a profuse sweat and relief to the pain in head and limbs.

"Dumb ague" is said to occur when there is fever and possibly sweating without a chill.

In the *masked forms* the paroxysm of chill and fever may be replaced by some other disturbance, such as neuralgia.

Symptoms in the *interval* between the paroxysms are not marked, but there is usually some sallowness of the skin and more or less enlargement of the spleen.

Diagnosis.—1. From *remittent* fever intermittent is diagnosed by the occurrence of *complete* intermissions in the latter.

2. From *pyæmia* it is diagnosed by the history of the case and by the serious condition of the patient between the paroxysms in pyæmia.

Prognosis.—The *prognosis* is nearly always favorable. If the chills occur at an earlier hour at each recurrence they are said to be "anticipating," and it is not a good indication; if they come at a later hour at each recurrence they are said to be "postponing," and it is a favorable sign.

Treatment.—*General prophylaxis.* Drainage and cultivation do more than any thing else to remove the favorable conditions for the development of the malarial germs.

Individual prophylaxis may be practiced by the administration of from two to five grains of quinine every morning before breakfast.

During the *cold stage* of a paroxysm morphia and chloroform to quiet the nervous system are often useful.

At the beginning of the *hot stage* antipyrine or antifebrine or phenacetine may be given to lower the temperature and hasten the occurrence of sweating. Pilocarpine has been used for the same purpose.

During the *sweating stage* no treatment is necessary.

In the *interval* between the paroxysm, quinine in full doses should be used. Opium increases its effect. Salicin, arsenic and nitric acid are also useful. The bowels should be opened by calomel.

In *chronic cases*, iron and cod liver oil are very beneficial.

REMITTENT FEVER.

Definition.—A malarial fever characterized by remissions, but not complete intermissions.

Symptoms.—Bilious fever; bilious remittent fever, &c.

Special Causes of this form of malarial disease—1. *Heat*; the severity of the malarial diseases usually increases with the temperature of the locality.

2. *Inflammatory complications*, which tend to make an ordinary intermittent fever assume a remittent type.

Morbid Anatomy.—1. The *blood* contains much free pigment from the destruction of the red corpuscles by the germs.

2. The *spleen* is enlarged from the action of the germs and the toxic principles formed by them.

3. The liver is congested and contains pigment.

4. The *stomach* and *bowels* are usually congested.

Symptoms.—1. *Digestive*, oppression in the epigastrium, nausea and vomiting; the tongue is at first pasty, later brown and dry; the bowels constipated at first, but often become loose afterwards. These symptoms are probably due to the action of the toxic principles formed.

2. The *nervous* symptoms consist of a chill, headache, back-ache and pain in the limbs, with extreme restlessness. The chill is not so violent as in intermittent fever. Delirium occurs later on.

3. The *temperature* rapidly rises to 105° or 106° , but after about twelve hours it falls two or three, or even four degrees; this is called the remission. As the disease advances the remissions which occur in the beginning at regular intervals become less and less marked.

4. The *circulatory* symptoms. The pulse is at first full and rapid, in consequence of the action of the heated blood on the muscular tissue of the heart and the nerves; later it becomes rapid and weak from degenerative changes.

5. The *general* symptoms are exhaustion and emaciation.

Bilious remittent fever is that form in which there is jaundice and large quantities of bile are vomited.

Diagnosis.—1. *Typhoid fever* differs from remittent fever in the gradual rise of temperature, and the early occurrence of diarrhoea in the former, and also in the more *continued* course of the fever.

2. *Yellow fever* differs from remittent fever in the occurrence of black vomit, and the much greater degree of jaundice in the former.

Prognosis.—The prognosis depends on—

1. The *type* of the epidemic.



2. The *locality*, the disease being more fatal in hot and marshy places.

3. The *complications*.

Duration.—There is no definite duration; the average length of an attack is about two weeks.

Treatment.—The *hygienic* treatment with respect to food stimulants and general regimen is like that of typhoid fever.

The indications in *remedial* treatment are—1. To *relieve symptoms* as—

(1.) Nausea by counter-irritants, morphia, chloroform, &c.

(2.) Constipation by calomel and enemata.

2. To *reduce temperature* by antipyretics.

3. To *destroy the germs* or neutralize the poison generated by them by quinine.

TYPHO-MALARIAL FEVER.

Definition.—A febrile affection characterized by a combination of the symptoms and morbid anatomy of typhoid fever and malarial affections, each being modified by the other. In its essential nature it is probably typhoid fever occurring in a person whose system is charged with the malarial poison.

Symptoms—Camp fever, malarial typhoid fever, continued malarial fever, &c.

Causes.—1. The *germ* of typhoid fever is probably the essential cause of typho-malarial fever, but it has not yet been found in the disease. The malarial germ is present in these cases also.

2. The *favorable conditions for development* are defective sewerage and defective drainage; in a word, moisture and filth. It is unnecessary to say more as to the causes, which are the same as those of typhoid fever and malarial affections combined.

Morbid Anatomy.—1. The *blood* is dark in color and coagulates badly.

2. *Parenchymatous** degeneration occurs in the heart, liver, kidneys and muscular tissue. The spleen is much enlarged. The explanation of these changes has been given in connection with typhoid fever.

3. The *intestines* are inflamed, especially in the neighborhood of the ileo-cæcal valve. Peyer's patches are enlarged and ulcerated, just as in typhoid fever, but the changes do not occur with the same regularity and order as in pure typhoid fever.

Complications.—*Inhalation pneumonia*, bronchitis, and laryngeal inflammations, as in pure typhoid fever.

Similar *glandular and intestinal* complications also occur.

Symptoms.—1. *Nervous.* A chill is common at the beginning of typho-malarial fever; headache and pain in the back and limbs occur just as in typhoid, and low muttering delirium, subsultus tendinum and other nervous symptoms occur in the later stages.

2. The *temperature* curve in malarial fever is different from that of pure typhoid. Instead of the gradual rise which is usual in the latter, the temperature often reaches 104° or 105° within forty-eight hours from the commencement of typho-malarial fever. There are frequently much more marked remissions also in this disease than in simple typhoid. The other symptoms are practically similar to those of uncomplicated typhoid fever and need not be repeated here.

Diagnosis.—1. From *simple typhoid* fever typho-malarial is distinguished by the much more rapid elevation of temperature, by the occurrence of remissions, and by the chill which often occurs at the commencement of the attack.

2. From *remittent fever* it is distinguished by the occurrence of diarrhœa and sometimes of the typhoid eruption.

Prognosis.—The mortality varies in different epidemics; it is usually about eight or ten per cent.

The *prognosis* is dependent on—

1. The *type* of the epidemic.
2. Bad *hygienic surroundings* and intemperate habits in the patient, which increases the mortality very much.
3. *Complications.*

Treatment.—The *prophylactic* treatment is the same as that of typhoid fever. The disinfection of the stools and bedding is especially important.

The *indications of remedial* treatment are—1. To *reduce temperature*; which may be accomplished by antipyretics, such as phenacetine, antipyrine, &c., and quinine, which is much more useful in this disease than in pure typhoid fever.

2. To *sustain strength* by the use of food and stimulants.

3. To *relieve symptoms*, such as nausea and vomiting, diarrhœa, headache, &c. The nausea is best relieved by small doses of morphine and codeine in conjunction with chloroform; these drugs lessen the irritability of the stomach. Diarrhœa is controlled by opium, bismuth, naphthaline and other astringents and antiseptics. Phenacetine and codeine give prompt relief to the headache and pain in the limbs.

4. *Specific treatment* directed to the malarial element is useful in those cases which are marked by decided remissions or in which there are other prominent features of malarial poisoning.

PERNICIOUS MALARIAL FEVER.

Definition.—Malarial affections so severe in character as to occasion death or very great danger.

Synonym.—Congestive chills.

Causes.—1. The *malarial poison*, as in other forms of malaria, is the *essential* cause.

2. *Heat.* The pernicious malarial fevers are very rare except in hot countries or in very hot weather.

3. *Certain unknown* conditions are also operative, for in the same locality pernicious malarial affections are much more common in some seasons than in others.

Morbid Anatomy.—The spleen and other internal organs show *extreme congestion*, pigmentation and sometimes infarctions.

The Varieties and Symptoms in each.—1. The *comatose*, in which during the hot stage the person becomes very drowsy and may sink into profound coma.

The pulse in this form is full and strong and slow and the respirations slow, the face flushed and pupils small.

The temperature often reaches 105° or even 107° .

2. The *delirious* form is characterized by wild delirium in the hot stage. The pulse is rapid and the respiration hurried.

3. In the *gastro-enteric* form there is profuse vomiting and purging. The pulse is rapid and feeble, and the extremities are cold. There is sometimes complete suppression of urine, and it is always scanty.

4. In the *algid* form, the cold stage persists, and the extremities become very cold, though the internal temperature is elevated. The pulse is rapid and feeble, and in bad cases disappears from the wrist.

5. The *icteric* form is characterized by the occurrence of jaundice, which comes on quite suddenly, usually in the hot stage; the pulse is often unusually slow in this form, and stupor and muttering delirium are common.

In the *hemorrhagic* form there is apt to be hemorrhage into the serous cavities and into the internal organs, especially the kidneys. The symptoms are very variable, depending on the seat and amount of the hemorrhage.

It should be remembered that *malarial hematuria* is not always pernicious in character.

It is not possible to explain the causes of these different forms.

The *icteric* form is probably due to a very great destruction of the red blood corpuscles; the *hemorrhagic* to profound changes in the walls of the blood vessels.

Diagnosis.—1 The *comatose* form is distinguished from *apoplexy* by the fever and the absence of hemiplegia, and by its gradual onset.

2. The *delirious* form is distinguished from *meningitis* by the chill which precedes it, and by the more rapid rise of temperature.

Prognosis.—The *prognosis* is usually unfavorable. It is based on the form—the *algid*, *icteric* and *gastro-enteric* being the most fatal—and on the locality—all cases being worse in a hot and marshy locality.

Treatment.—During a *paroxysm* morphia and chloroform are very useful; antipyrine and pilocarpine have also been used with benefit in the *comatose* and *delirious* forms, and also in the *algid*.

In the *interval*, and sometimes even during a *paroxysm*, *quinine* should be given hypodermically, in doses of about three grains every two or three hours.

CHRONIC MALARIAL TOXAEMIA.

Causes.—Chronic malarial toxæmia may occur: 1. In those persons who have long been residents of a malarial region, but who have never had any acute manifestations of the disease.

2. As a sequel of the acute attacks.

Morbid Anatomy.—The *morbid anatomy* is similar to that of the acute attacks except that it is greater in degree. The spleen is more enlarged, the liver and kidneys are apt to present evidences of degeneration. The heart is usually softened and is flabby.

Symptoms.—The symptoms vary very greatly in different cases. 1. The most common *nervous* symptoms are neuralgia in different parts of the body, headache, sleeplessness, depression of spirits and occasionally actual melancholy. Paralysis occurs occasionally.

2. The *digestive* symptoms are very variable. Sometimes diarrhoea occurs, the actions being light colored and pasty. Sometimes on the contrary there is constipation. The appetite is nearly always impaired. Tenderness over the liver and spleen is usually observed.

3. The *respiratory* symptoms are "shortness of breath," which is due to the diminution in the number of red corpuscles, and sometimes bronchitis and cough.

4. The *circulatory* symptoms are weakness of the heart's action and palpitation, both of which are due to defective nutrition.

5. The *general* symptoms are pallor, from destruction of red corpuscles, debility from degeneration of the heart and muscular tissue, and very often *dropsy* from weakness of the heart and degenerative changes in the vascular walls.

Diagnosis.—The *diagnosis* is based on—1. The enlarged spleen.

2. The remissions which usually occur.
3. The presence of pigment in the blood.
4. The effect of specific treatment with quinine.

Prognosis.—The *prognosis* depends on the morbid changes and symptoms. If the spleen is greatly enlarged and tender, and there is tenderness and enlargement of the liver, and dropsy, it is unfavorable.

Treatment.—The *treatment* consists in change of residence, the wearing of flannel to promote the action of the skin, and the administration of quinine, iron, cod-liver oil and arsenic. Warburg's tincture often gives better results than anything else.

MUMPS.

(Parotitis.)

Definition and Frequency.—An acute, infectious and contagious disease characterized by swelling of the parotid glands. It is an exceedingly common affection.

Causes.—1. A *germ* which has not been isolated.

2. *Age.* It is far more common in children than in adults, but may occur at any age. Young infants usually escape.

3. The *avenue of introduction* is probably the respiratory mucous membrane.

4. The *medium of contagion*, the atmosphere, in most cases if not in all.

5. Immunity is usually conferred by one attack.

6. The *period of incubation* is from 12 to 21 days.

Morbid Anatomy.—The parotid glands—one or both—are hyperæmic and infiltrated with serum.

Symptoms—A. *Prodromic* symptoms, such as malaise, slight fever and headache, are frequent.

B. Symptoms of *developed* attack.

1. *Glandular.* One or both parotid glands become swollen, tender and painful. The swelling has no tendency to suppurate.

2. The temperature is elevated to 102° or 103° generally, but may rise to 106° .

3. The *pulse* is quickened and *appetite* lost.

Diagnosis.—The *diagnosis* is based on the swelling of the carotid, without pyæmic symptoms.

Prognosis.—The *prognosis* is nearly always favorable.

Complications.—1. *Glandular.* The *testicles* in males, or the *breasts* or *ovaries* in women, may become greatly inflamed and tender.

2. Serious *cerebral* symptoms, violent headache, stupor or delirium may occur, but usually pass off in a few days or, possibly, a few hours.

Treatment.—*Treatment* is of little value. The indications are to give relief from pain and to lessen the severity of complications.

Rubbing with camphorated oil is the best external treatment. The bromides and phenacetine may be given in case cerebral symptoms arise.

CHAPTER III.

DISEASES OF THE RESPIRATORY ORGANS.

ACUTE CORYZA.

Definition.—An acute catarrhal inflammation of the mucous membrane of the nose.

Synonyms.—Acute nasal catarrh, cold in the head, &c.

Causes.—1. *Sudden chilling* of the body when overheated; the way in which this acts is obscure.

2. *Injuries*, whether *mechanical*, as cuts or blows, or inspissated mucous; or *chemical*, as from the inhalation of certain irritating vapors or from the elimination of certain drugs taken internally, such as iodine.

3. The *exanthemata*, especially measles.

Morbid Anatomy.—1. *Redness* and *swelling* of the mucous membrane and a catarrhal exudate. There is also distension of the erectile tissue over the turbinated bones.

Symptoms.—1. *Nervous*. Sometimes there is a chill or chilly sensations, some headache, a feeling of dullness and sometimes pain in the back and limbs.

Sneezing almost always occurs from the increased reflex irritability of the nerves of the nasal mucous membrane.

2. *Secretory*. At first there is a thin secretion, from the stimulation of the mucous follicles by the increased flow of blood; later on a catarrhal exudate.

3. *Mechanical*. There is stopping up of the nose, and so-called nasal speech from the swelling of the mucous membrane over the turbinated bones.

Prognosis.—Recovery nearly always occurs in a few days. Rarely chronic coryza results.

In the case of infants, the interference with sucking may cause debility and even death.

Treatment.—The first indication is to *increase* the amount of blood *in the skin* and in that way cause a collateral anæmia of the nasal mucous membrane. For this purpose warm baths, hot drinks, quinine, Dover's powders, jaborandi and antipyrine may be used with great advantage.

The *local* treatment consists in *inhalation* of tincture of iodine and ammonia, a *spray* of borax and bicarbonate of sodium, and the

use of a snuff of cocaine, antipyrine, boracic acid and bismuth. These agents seem to destroy the germs which lodge in the nasal mucous membrane and help to keep up the inflammation. Antipyrine also causes contraction of the blood vessels and cocaine relieves pain.

CHRONIC NASAL CATARRH.

Definition.—A chronic catarrhal inflammation of the nasal mucous membrane.

Synonyms.—Catarrh, *ozæna*.

Causes.—1. *Acute coryza*.

2. *Atmospheric condition*, such as dampness and the presence of irritating vapors, such as tobacco smoke and the fumes generated in certain manufacturing operations.

3. *Syphilis, tuberculosis and scrofula*. These are very common causes of chronic nasal catarrh.

Morbid Anatomy of—1. *Hypertrophic form*. There is swelling and redness of the mucous membrane and hypertrophy of the tissues over the turbinated bones.

2. *Atrophic form*. In this form there is *atrophy* of the mucous membrane and the tissues beneath. The mucous follicles are destroyed and even the bony tissue is thinner than natural.

Symptoms.—1. *Secretory*. There is, in the hypertrophic form especially, a more or less copious *secretion* of yellowish or greenish muco-pus, which frequently dries and forms *crusts* in the nose. The discharge is often offensive from the decomposition set up by germs.

2. *Respiratory*. In the hypertrophic form there is often some obstruction to *nasal* respiration; in the atrophic form, on the other hand, the air passes through the nose very easily.

Physical Signs.—On examination with the rhinoscope the tissues may be seen thickened and red in the hypertrophic form, or pale and dry and atrophied in the atrophic form.

Diagnosis.—The *diagnosis* is not difficult. It is important to look for the evidences of scrofula or tubercle or syphilis, to determine whether these conditions cause the catarrh.

Prognosis.—The *prognosis* as to *life* is good; as to *recovery* it is not very good, unless the disease is slight and is due to syphilis.

Treatment.—1. *General*. To *improve* the *general health* by tonics, &c. To reside in a *dry* country is of more importance than anything else. To treat syphilis when present.

2. *Local.* To remove crusts by sprays containing bicarbonate of soda and by the application of vaseline to soften them.

To lessen fetor by carbolic acid or permanganate of potash solutions used with an atomizer.

To remove hypertrophic tissue by caustics or the knife. The galvano-cautery is very useful, for it prevents hemorrhage.

NOSE BLEED OR EPISTAXIS.

Causes.—1. *Injuries* which cause rupture of the vessels.

2. *Hemorrhagic diathesis.*

3. Certain forms of *heart disease* in which the vascular walls are weakened and the heart's impulse is increased.

4. *Purpura, leukæmia, &c*, in which there are changes in the blood and disease of the walls of the vessels also.

Treatment.—1. *Quiet*, to allow a coagulum to form in the injured vessel.

2. *Antipyrine*, which causes contraction of the blood vessels. It should be used as an injection into the nose or as a snuff.

3. *Monsel's Solution.*—The nose may be plugged with absorbent cotton soaked in dilute Monsel's Solution.

4. *Plugging* of the anterior nares and of the posterior nares by means of Bellocq's cannula.

5. *Ergot* is sometimes given internally, but it is of doubtful value.

ACUTE CATARRHAL LARYNGITIS.

Causes.—1. *Constitutional predisposition* has an evident influence in causing acute laryngeal catarrh, some persons being far more liable to it than others.

2. *Chilling* of the body when overheated.

3. *Mechanical* or *chemical* irritants, as from the inhalation of irritating powders or vapors.

4. It occurs also in measles and other of the *acute exanthemata*, typhus, &c.

Morbid Anatomy.—1 *Redness* and some swelling.

2. *Exudation*, which is small in quantity and serous in character at first; later becoming sero-purulent.

3. *Infiltration* of the *sub-mucous tissue* with serum occurs in severe cases.

Symptoms.—1. The *voice* is affected, there being more or less hoarseness, or in severe cases complete aphonia.

2. *Respiration* is not affected unless there is sub-mucous infiltra-

tion, when it is seriously interfered with. Cough of a ringing and spasmodic character is often present from the increased reflex irritability.

3. *Pain* is rarely present, but there may be a slight feeling of soreness.

4. The *secretion* is slight because the laryngeal mucous membrane contains few mucous glands.

Physical Signs.—On *laryngoscopic examination* the vocal cords are observed to be reddened; there is some muco-purulent secretion on them, and the movements of the muscles are impaired.

False Croup, which occurs in children, is due to a *reflex spasm* of the larynx. The acute laryngitis increases the irritability of the sensory nerves, and this causes reflex spasm of the larynx.

The symptoms of false croup are a *ringing cough*, from the vibration of the tense vocal cords, and dyspnœa, from the spasmodic narrowing of the wind-pipe.

Diagnosis.—From *diphtheria* acute laryngitis is diagnosed by the absence of any false membrane in the latter.

From *hysterical laryngeal spasm*, by the suddenness of the onset of hysterical attacks, and by the absence of evidences of inflammation.

Prognosis.—The *prognosis* is usually very favorable.

It is sometimes serious in young children, and there is especial danger in them of extension to the bronchi. When due to the inhalation of irritating vapors, such as steam, it may cause death from œdema.

Treatment.—1. To *lessen congestion* by dilating the vessels of the skin, is the first indication.

2. To *promote secretion* by the use of steam inhalations, to which compound tincture of benzoin may be added.

3. To *facilitate the entrance of air*, when œdema is present, by means of scarification or tracheotomy.

CHRONIC LARYNGITIS.

Definition.—Chronic catarrhal inflammation of the larynx.

Causes.—1. *An acute attack.*

2. *Occupation*; persons who do much public speaking are especially liable to it; hence it is sometimes called clergyman's sore throat; singers are also apt to suffer from the disease.

Persons who work where there are irritating vapors or fumes are liable to it.

3. *Dissipation* is a very potent cause, it being especially common among drunkards.

Morbid Anatomy.—1. *Redness, swelling* and sometimes *ulceration* of the mucous membrane of the larynx.

Irregular swelling and contraction are caused by the formation of connective tissue in the mucous and sub-mucous tissue.

Symptoms.—1. The *voice* is affected to a greater less degree. Hoarseness is nearly always present, and even when it is not apparent in ordinary conversation, it is impossible to strike the high notes in singing.

2. The *respiration* is not affected, as a general thing. There is some *expectoration* of tough mucus of a yellowish color, usually, but the amount expectorated is small.

The *nervous* symptoms are *cough*, which is not often troublesome, and a feeling of weakness and soreness of the throat, rather than of actual pain.

Physical Signs.—On *laryngoscopic examination* the vocal cords present the appearances mentioned under Morbid Anatomy.

Diagnosis.—The *diagnosis* is based on the history of the case and the results of the laryngoscopic examination. It has to be distinguished especially from tuberculosis and syphilitic laryngitis, or tumors of the larynx.

Prognosis.—The *prognosis* as to *life* is very good ; as to *recovery*, it must be guarded ; it will depend very much on the climatic and hygienic conditions.

Treatment.—A. *Hygienic.* Rest to the voice, the avoidance of irritants, such as smoking, drinking, &c. A warm and equable climate are of the utmost importance.

B. *Remedial* treatment consists in

1. *Inhalations* of weak solutions of tannic acid, alum, boracic acid, &c.

2. *Applications* to the larynx, with the *brush*, of solutions of nitrate of silver, carbolic acid, iodoform, &c.

3. The *insufflation* of *powder*, such as boracic acid, iodoform, bismuth, sulphur, &c.

4. *Constitutional treatment* with the object of causing a collateral hyperæmia of the abdominal organs and a consequent anæmia of the organs in the thorax. The sulphur waters, and others which act on the bowels, have been found of service.

LARYNGEAL PERICHONDritis.

Definition.—An acute inflammation of the peri-chondrium.

Causes.—1. *Tuberculosis, syphilis, cancer, &c.*

2. Rarely the *acute infectious diseases*, especially small-pox and typhus.

Morbid Anatomy.—1. *Seat* in the cartilages of the larynx.

2. The *exudate* is purulent and collects between the perichondrium and the cartilage.

3. The *cartilage* is *destroyed* and discharged with the pus.

Symptoms and Signs.—Those of *stenosis* from swelling of the larynx and consequent narrowing of its calibre. *Pain* is also present and is often severe.

Diagnosis is made by the history of the case and laryngoscopic signs.

Prognosis is bad. If recovery occurs there is apt to be permanent narrowing of the larynx.

Treatment.—*Tracheotomy*, to admit air, and then the removal of pus from the abscess.

ŒDEMA OF THE GLOTTIS.

Definition.—Infiltration of the sub-mucous tissue of the larynx with serous fluid.

Causes.—1. *Acute laryngitis*.

2. *Perichondritis*.

3. *Dropsy*, from heart disease or Bright's.

Symptoms.—*Dyspnoea* is the prominent and essential symptom.

Treatment.—1. *Incisions*, to let out the fluid.

2. *Tracheotomy*, to permit the entrance of air.

SPASM OF THE GLOTTIS.

Causes.—1. *Age and sex*, most common in boys under three years old.

2. *Rickets* predisposes to it: the reason is not known.

Symptoms.—1. *Sudden dyspnoea*, from closure of the glottis.

2. *Convulsions* sometimes, from retention of carbon dioxide in the blood.

Prognosis.—Usually good so far as the attack itself is concerned.

Treatment.—1. To *improve the general health*.

2. *Chloroform* during paroxysm.

TUBERCULOSIS OF THE LARYNX.

Causes.—1. The *bacillus tuberculosis* is the essential cause.

2. It may be primary, but is usually *secondary* to tuberculosis of the lungs.

Morbid Anatomy.—1. *Redness* of the cords with little whitish elevations (tubercle.)

2. Later on, *ulceration* of the cord, the ulcers being caseous and showing no disposition to heal.

Symptoms.—1. *Hoarseness* or complete aphonia.

2. *Cough*, often troublesome.

3. Difficult and painful *deglutition* from ulceration and irritability of the epiglottis.

4. *Weakness*, *pallor*, night sweats, &c., as in tuberculosis of other organs.

Diagnosis based on—1. The presence of tuberculosis elsewhere.

2. The character of the ulcers.

Prognosis.—Uniformly unfavorable.

Treatment.—1. To *improve* the *general health* and sustain strength.

2. To *apply germicides*, such as iodoform, to the ulcers. This treatment is of very doubtful efficacy.

3. To *facilitate deglutition*. The use of a spray of cocaine (10 per cent) just before eating gives very great relief.

SYPHILIS OF THE LARYNX.

Period of Occurrence.—*Secondary*.

Morbid Anatomy.—1. *Redness and swelling*, usually in patches.

2. *Ulceration*, the ulcers being inclined to heal on one side and extend on another.

Symptoms.—1. The *voice* is seriously affected; hoarseness is nearly always present and there may be complete aphonia.

2. *De-glutition* is painful and difficult.

3. *Cough* is present, and in late stages more or less *dyspnoea* is common from cicatricial contraction.

Diagnosis based on—1. The history of the case.

2. The character of the ulcers.

Prognosis.—Favorable in the early stages; later, stenosis is apt to occur.

Treatment.—The *iodides* in large doses and mercury.

PARALYSIS OF THE LARYNGEAL MUSCLES.

Causes.—1. *Central*, as degeneration of the nerve centres in bulbar paralysis.

2. *Peripheral*, from pressure on the nerve trunks by tumors or degeneration of the nerves, as in diphtheria.

3. Changes in the *muscles themselves*, as in certain degenerations.

4. *Functional*, as in hysteria.

Symptoms.—1. The *voice* is always affected and often there is complete aphonia.

2. *Respiration* may be interfered with, but it is usually unaffected.

Diagnosis.—The *diagnosis* is based on the history of the case, and the results of a laryngoscopic examination.

Prognosis.—The *prognosis* depends altogether upon the cause. If the pressure can be removed from the nerve, for instance, it is favorable. In hysterical cases it is favorable.

Treatment.—1. To *remove the cause*.

2. To *stimulate the nerves* by electricity, strychnia and other tonics.

PHYSICAL DIAGNOSIS OF AFFECTIONS OF THE RESPIRATORY ORGANS.

Divisions of the Chest.—A. *Anterior*.—1. Supra-clavicular, above the clavicle.

2. Infra-clavicular, from the clavicle to the third rib.

3. Mammary, from the third to the seventh rib.

4. Infra-mammary, from the 7th rib to the edge of the thorax.

B. *Lateral*.—1. Axillary, from the arm pit to a line connecting the lower angle of the scapula with the lower border of the infra-clavicular region.

2. Sub-axillary, from the axillary to the twelfth rib.

C. *Posterior*.—1. Supra-scapular, above the spine of the scapula.

2. Scapular, underneath the scapula.

3. Infra-scapular, from the lower angle of the scapula to the twelfth rib.

4. Inter-scapula, between the scapulae.

Methods of Physical Diagnosis.—1. Inspection.

2. Palpation.

3. Percussion.

4. Auscultation

Inspection—*Methods*. The patient must be stripped to the waist. The physician should stand in front of the person or direct

ly behind him. The person to be examined should stand erect with the arms falling loosely at the sides.

Objects of Inspection. To determine (1) the size of the chest, (2) the shape of the chest, (3) the movements of the chest, (4) the relative size and movements of the two sides.

Changes in Disease.—The *size* of the chest may be *greater* than normal from an excessive amount of air in the air cells (emphysema,) from muscular development, and possibly from dropsy.

The *shape* of the chest is altered in certain diseases, as emphysema, when it is barrel-shaped, and as a result of pleurisy with effusion on one side or with retraction of the chest walls from adhesions and shrinking of the lung.

The *movements* of the chest are very frequently changed by disease. If respiration causes *pain* on one side, as in pleurisy, or pleurodynia, the movements on that side are less extensive than normal. If there is *effusion* into the *pleural* sack, the movements are greatly lessened or are absent.

Palpation.—*Method.*—To practice palpation the hands should be placed upon the chest walls.

Objects. The objects of palpation are to determine the size, shape and movements of the chest and also the character of the *vocal fremitus* and in certain diseased conditions the *rhoncal fremitus* also.

The *vocal fremitus* is the peculiar thrill imparted to the hand placed upon the chest of a person who is talking.

Its degree depends upon (1) the thickness of the chest walls—thick walls lessen the fremitus—and (2) the *pitch* of the voice; it is greater when the voice is low in pitch than when it is high.

Changes in Disease.—The *vocal fremitus* may be (1) increased or (2) diminished or lost in diseased conditions. It is *increased* whenever the lung tissue is *solidified*, as in pneumonia or tubercular consolidation.

It is *diminished* or *lost* (1) when there is a layer of fluid between the lung and the chest wall, as in pleurisy with effusion; (2) when the bronchus leading to the portion of lung under the examining hand is stopped up.

Rhoncal fremitus is never present in health. It is due to the *vibration* occasioned by narrowing of the bronchial tubes or by the presence of mucus in the tubes.

Percussion Methods.—There have been two methods of percussion practiced—(1) the *immediate*, in which the chest is struck directly, and (2) the *mediate*, in which some other body, usually the fingers of one hand, are placed on the chest and struck with the percussing fingers. A little hammer and a piece of rubber or ivory may be used in place of the fingers.

The mediate method is nearly always adopted now.

Object.—The *object* of percussion is to determine the relative

amounts of air and solid or liquid matter in the lung or chest cavity.

The sounds elicited on percussion are to be considered with respect to (1) *quality*, (2) *pitch*, (3) *duration*, (4) *intensity*.

The percussion sounds in different regions of the chest depend in the healthy person upon (1) the difference in thickness of the thoracic walls in different persons and at different parts of the chest, and (2) upon the character of the underlying tissues or organs.

The percussion sounds are clearer in the axillary and sub-clavicular regions, as a rule, than elsewhere, because the chest walls are thinner there. There is more resonance in the left sub-clavicular region than in the right, because the large bronchus on the right side lessens the amount of pulmonary tissue. There is absence, or great diminution, of resonance over the region of the heart, and also below the sixth rib on the right side, because the liver lies beneath.

Changes in the percussion sounds produced by disease.—1. *Flatness*, or an absence of all resonance is nearly always due to an accumulation of fluid in the pleural cavity, or a tumor there.

2. *Dullness*, or a diminution of resonance is due to a relative or actual diminution in the quantity of air in the lung. It is consequently observed in *pneumonia*, *tubercular infiltration*, and in any other condition in which the amount of air is lessened.

3. *Tympanitic* resonance is due to an actual or relative increase in the amount of air in the lungs, as in *emphysema*, and in certain cases where there is a *cavity* in the lungs.

3. *Amphoric* resonance is characterized by its *metallic* character; it is obtained by percussing over *cavities with firm, elastic walls*.

5. The *cracked-metal sound* is produced by percussing over cavities which communicate with a bronchus by a small opening.

Auscultation.—*Methods.* By *auscultation* is meant the listening to the sounds in the chest. There are two methods, (1) the *immediate* when the ear is applied directly to the chest walls, and (2) the *mediate* when a stethoscope is employed.

Sounds over the *healthy* chest.

The respiratory or *vesicular murmur* is the sound produced by the entrance of air into the air vesicles. The *thinner* the chest walls and the *deeper* the *inspirations* the louder the vesicular murmur is. Its intensity or loudness varies greatly in different persons. It is heard most distinctly in the sub-clavicular and axillary regions, because the chest walls are thinner there.

Over the upper part of the sternum and in some children underneath the right clavicle and between the upper ends of the scapulæ a sound is heard like blowing through a tube. This is known as



bronchial or *tubular* breathing and is owing to the proximity of the large tubes to the walls of the chest at these points.

The vesicular murmur is heard during *inspiration*. The expiratory sound is much shorter than the inspiratory and rather more blowing in character. *Inspiration* is due to the action of the powerful muscles; *expiration* is due chiefly to the elasticity of the lungs.

Changes in the vesicular murmur in disease.

Alterations in intensity. 1. *Increased or puerile respiration* is usually compensatory; for example, if one lung is solidified or compressed the sounds over the other lung are increased.

2. *Diminished or feeble respiration* may be due (1) to some obstruction to the entrance of air into the lungs as the plugging of a bronchus or weakness of the inspiratory muscles, or (2) to some hindrance to the transmission of sound to the ear such as a thin layer of fluid in the pleural cavity, or (3) to a loss of elasticity of the air cells which prevents the discharge of air from the air cells prior to their refilling with air.

3. *Absence of respiratory sound* is due to the *complete plugging* of a bronchus or to a thick layer of fluid between the lungs and chest walls.

Alterations in rhythm.—1. *Jerky respiration* is due to irregular dilatation of the tubes and air cells; it has little clinical significance

2. Change in the *relative length of inspiration and expiration* is of great practical importance. *Prolonged expiration* is an evidence of a loss of elasticity of the walls of the air vesicles, and this loss of elasticity may be occasioned by *over-distension* of the air cells as in *emphysema* or deposits which impair their contractile power as tubercle.

Alteration in character. Harsh, *broncho-vesicular*, respiration is a mixture of the vesicular murmur with bronchial respiration. It shows a diminution in the relative amount of air. When heard at the apex of the lung it is very suggestive of tuberculosis.

Bronchial Respiration is heard in health over the upper part of the sternum and in some persons, especially children, under the right clavicle and between the spines of the scapulæ, because large tubes are in close proximity to the chest walls at these points.

Bronchial respiration, or tubular respiration, if heard elsewhere is an evidence of *consolidation* of the *lung tissue* beneath. It is observed, therefore, in *pneumonia*, *tubercular infiltration* and *collapse* of the lung.

Bronchial respiration may be absent over consolidated lung tissue, however, (1) if the bronchus leading to the consolidated lung is stopped up, (2) if there is a layer of fluid between the lung and the chest walls, as in pleurisy with effusion, or (3) if the consolidated lung is central and surrounded by healthy tissue.

Amphoric Respiration resembles the sound produced by blowing over the mouth of a bottle.

It is heard over cavities with tense, elastic walls and is due to the echo from these walls.

New or Adventitious Sounds are those which are heard only in disease.

They are *râles* or *rattling* sounds which are produced in the bronchial tubes, in the air vesicles or in cavities, and *friction sounds*, which are produced by the rubbing against each other of the costal and pulmonary pleura which have been roughened by inflammatory exudate.

Râles may be *dry* or *moist*. *Dry râles* are usually caused by narrowing of the tube by spasmodic contraction, or swelling, or a piece of tough mucus sticking to the side.

Moist râles are due to the passage of air through mucus.

Dry râles, formed in *large* tubes, are *low-pitched* or sonorous; those formed in *small* tubes are *high-pitched*, or sibilant, or whistling.

Moist râles, too, vary in size; those found in *large* tubes are *large*, *bubbling* or mucous; those in *small* tubes are much smaller and are called *sub-crepitant*. They vary in size.

Vesicular râles are those which are formed in the air vesicles; the sound resembles that caused by rubbing the hair between the two fingers. It is called *crepitation* and is probably due to the pulling apart of the sticky walls of the pulmonary alveoli during inspiration.

Crackling is the same thing as crepitation, except that the sounds are few in number.

Two kinds of *râles* may be heard over cavities—(1) *hollow bubbling* or gurgling, when the cavity has *flabby* walls, and (2) *metallic*, when the cavity has *tense, elastic* walls.

Friction sounds are caused by the rubbing against each other of two pleural surfaces, roughened by inflammatory exudate. As a rule, the sound usually seems to come from a point very close to the ear.

The following table is taken from DaCosta's Medical Diagnosis :

Bronchial Râles	{ Dry or Vibrating { 1. Low pitched or sonorous in large tubes. { 2. High pitched or sibilant in small tubes. { Moist. { 1. Large bubbling, or mucous in large tubes. { 2. Small bubbling, or sub-crepitant, in small tubes.
Vesicular Râles	{ 1. Crepitation, due to the separation of the walls of the alveoli in inspiration. { 2. Crackling—a few crepitant râles.
Râles of Cavities	{ 1. Hollow bubbling or gurgling in large cavities with flabby walls. { 2. Metallic in cavities with tense, elastic walls.

AFFECTIONS OF THE BRONCHIAL TUBES.

ACUTE BRONCHITIS.

Definition.—An acute catarrhal inflammation of the mucous membrane of the larger bronchi.

Synonym.—Cold in the chest.

Causes.—1. *Constitutional predisposition* is one of the causes of acute bronchitis; its action cannot be explained.

2. *Cold and dampness* are common causes, probably because they cause a contraction of the blood vessels of the skin and consequently a collateral hyperæmia of the internal organs.

3. *Mechanical and chemical* irritants, such as the inhalation of ammonia, or of septic matters from the mouth, sometimes occasion the disease.

4. It is a frequent attendant of *certain other diseases*, such as measles, whooping cough, &c.

Morbid Anatomy.—1. The *seat* of the disease is in the *larger* bronchi.

2. The *changes in the mucous membrane* are swelling and redness.

3. The *exudate* is at first serous or mucous, and is scant; later it becomes more abundant and still later it becomes purulent in character.

Symptoms.—1. *Respiratory.* The only respiratory symptoms as a rule are (1) *cough* and (2) *expectoration*.

The *cough* is at first tight and often painful; it is often quite troublesome.

The *expectoration* is at first scant and clear; a little later much more abundant, clear and frothy, and still later thick and purulent.

2. The *nervous* symptoms are not marked; a feeling of constriction of the chest is present in the early stages and headache is often present.

3. The *temperature* is rarely elevated to any appreciable extent.

Physical Signs.—On *auscultation*, *sonorous* and *sibilant râles* are often heard from the narrowing of the tubes by thick mucus and *moist râles* are also heard, due to the passage of air through the mucus in the tubes. The *râles* vary in size because tubes of different sizes are involved; sometimes, when only large tubes are involved, there are no *râles*.

The **Diagnosis** is not difficult; it is based on the cough, expectoration and physical signs.

The **Prognosis** is almost invariably good; there is a possibili-

ty that the disease may become chronic or that it may pass into capillary bronchitis.

Treatment—The *indications* and mode of fulfilling them are as follows:

1. To *relieve inflammation* by causing hyperæmia of the skin by hot baths, Dover's powders, &c., and by acting on the bowels.
2. To *promote expectoration* in the early stages by the inhalation of steam, the internal administration of ipecac, &c.
3. To *check* expectoration, when profuse, by the terebinthines, such as turpentine, cubebs, &c., and by muriate of ammonia.
4. To *relieve cough* and *soreness* by opiates, chloroform and other sedatives.

CHRONIC BRONCHITIS.

Definition.—A chronic inflammation of the bronchial tubes, leading in many cases to increased formation of connective tissue and to cylindrical dilatation of some of the tubes.

Causes.—1. *Age.* The disease is much more common in elderly people than in younger persons, but it may occur at any age.

2. It occasionally follows an *acute attack*.

3. *Mechanical* and *chemical* irritants, if inhaled, may induce the disease.

4. It is often *secondary* to chronic *cardiac*, *renal* or *pulmonary* disease. These diseases act by interfering with the proper circulation of blood through the lungs.

Morbid Anatomy.—1. The *mucous membrane* is congested and usually thickened, but in very chronic cases it may become smooth and thin.

2. The *tubes* are often dilated in a fusiform manner from the softening of the tissues and the pressure of the secretions.

There is an *increase of connective tissue* in the peri-bronchial tubes, from the conversion of the white blood cells into connective tissue.

Symptoms.—1. *Respiratory.* *Cough* is present in nearly all cases, and is often very harrassing; it is usually much worse in winter than in summer.

The *expectoration* varies in different cases; sometimes it is very scant and tough; sometimes profuse and yellow, and sometimes exceedingly copious and watery. In some cases, especially where the tubes are dilated, the sputa are exceedingly offensive ("fetid bronchitis").

Dyspnea is occasionally present, but is usually due to some complication.

2. *Circulatory.* The circulatory symptoms in chronic bronchitis are not marked, unless there is some obstruction to the flow of

Physical Signs.—On *inspection* there are no marked characteristics in uncomplicated cases.

On *palpation* there is no change in the *vocal* fremitus, but there may be some *rhoncal* fremitus.

On *percussion* there is no appreciable change.

On *auscultation*, as a rule, râles of different size and character are heard in consequence of the presence of mucus in the tubes.

Diagnosis.—Chronic bronchitis is diagnosed from *pulmonary tuberculosis* by (1) the absence of dullness on percussion, (2) the absence, as a rule, of any elevation of temperature and (3) the absence from the sputa of the bacilli of tuberculosis.

Complications.—1. *Emphysema*, to which the bronchitis is usually secondary.

2. *Bronchiectasis*, which is secondary to the bronchitis and is caused by the accumulation of secretion in the tubes, which are softened by inflammation.

3. *Renal*, which usually precede the bronchitis and stand in a causative relation to it.

4. *Cardiac*. In old cases of chronic bronchitis hypertrophy and dilatation of the right side of the heart occur from the obstruction to the flow of blood through the lungs.

Sequelæ.—1. *Pulmonary*. *Bronchiectasis* and more or less *peribronchitis*: the manner in which they are produced has already been described.

2. The *cardiac* have been described under complications.

Treatment.—The *indications* of treatment are: 1. To *remove the cause*. A change of climate to a mild and warm one in winter is of great importance. To stimulate the action of the kidneys is also important.

To *improve the general health* by tonics and cod liver oil is very important.

3. To *check expectoration* by the terebinthinate preparations, muriate of ammonia, &c.

CAPILLARY BRONCHITIS.

Definition.—A catarrhal inflammation of the *smaller bronchi*.

Frequency.—The disease is quite a common one, especially in old persons and young children.

Causes.—1. *Age*. The disease is far more common in old persons and young children than in vigorous adults, because the latter can more readily expel the mucus from the large tubes, and hence inflammation is less liable to extend to the smaller ones.

2. Certain *other diseases*, such as typhoid fever, measles, &c., which induce debility and permit the inhalation of noxious matters from the mouth or throat.

3. *Exposure* to cold and dampness, especially when the body is overheated, which causes contraction of the blood vessels in the skin and a sudden congestion of internal organs.

Morbid Anatomy.—1. *Seat.* In capillary bronchitis the inflammation is situated in the *smaller tubes*.

2. *Changes* in the *mucous* and *sub-mucous* coats. The mucous membrane is reddened and swollen; the cells lining it undergo fatty degeneration and peel off; there are also leucocytes in the membrane in considerable number. The sub-mucous tissue is more or less infiltrated with leucocytes.

3. *Character* of the *exudate*. The exudate consists of serous fluid or white blood corpuscles, which are mixed with the mucous secretion and with degenerated epithelial cells.

Symptoms.—1. *Respiratory.* The most striking symptom is *dyspnea*, which is due to the swelling of the mucous membrane of the smaller tubes and the consequent obstruction to the entrance of air. *Cough* is usually troublesome, and is due to the increased irritability of the terminal filaments of the sensory nerves and to the presence of mucus and exudate in the tubes.

Expectoration is usually scanty, because patients with this disease have not sufficient strength to expel the contents of the tubes.

2. *Circulatory.* The pulse is rapid and very feeble in consequence of the exhaustion and the effort of the heart to force the blood on through the vessels. *Cyanosis* is often present because the swelling of the tubes prevents the entrance of air into the lungs and the blood remains venous.

3. *Nervous.* The retention of the carbon dioxide in the blood causes restlessness, which in severe cases is followed by gradually increasing stupor and finally coma. Sometimes, especially in children, the carbon dioxide causes convulsions.

4. *Temperature.* The temperature is not usually much elevated unless broncho-pneumonia is present as a complication. Sometimes in elderly persons it is sub-normal.

5. *Cutaneous.* Besides the cyanosis, sweating is frequently observed, especially about the head.

Physical Signs.—1. *Inspection.* Both inspiration and expiration are rendered difficult by the swelling of the mucous membrane of the bronchioles.

2. On *palpation* there is often no change, though the fremitus may be rendered more feeble than natural by the plugging of a tube.

3. In *percussion* there is often no change, but sometimes inspiration being more powerful than expiration, the air cells become

over-distended, and then there is increased resonance on percussion.

4. On *auscultation* sub-crepitant râles are heard in consequence of the narrowing of the smaller tubes by mucus or the swelling of the mucous membrane.

Diagnosis.—It is diagnosed from (1) *bronchitis* of the *larger tubes* by the greater dyspnœa, the cyanosis and the sub-crepitant râles; from (2) *pneumonia* by the absence of bronchial respiration and dullness on percussion.

Complications.—1. *Collapse of the lung* is very common; it is due to the closure of a bronchus and the absorption of the air in the corresponding part of the lung.

2. *Broncho pneumonia* is also common; it is due to the extension of the inflammation from the bronchioles into the air vesicles.

Prognosis.—The *prognosis* should be guarded in all cases. The more debilitated the patient the greater the danger.

Treatment.—The indications are—1. To *reduce inflammation*, by causing collateral hyperæmia of the skin by means of poultices, turpentine stupes, &c., applied to the chest, and by the administration of ipecac, if the exhaustion is not too great.

2. To *promote expectoration*, carbonate of ammonia is the best remedy because it liquefies the sputa and also acts as a stimulant. Ipecac or apomorphia may be used, if the prostration is not too great.

3. To *sustain strength* by nourishing food and stimulants, such as milk punch, beef tea, wine and carbonate of ammonia. Musk and strychnine are also useful stimulants.

4. To *furnish oxygen* to the blood by allowing the patient to inhale oxygen gas.

CROUPOUS BRONCHITIS.

Definition.—An inflammation of the bronchial tubes in which the exudate is fibrinous in character. The disease is very rare.

Causes.—The *causes* are practically unknown. The disease occurs most frequently in middle aged men.

Morbid Anatomy.—The *morbid anatomy* is similar to that of simple bronchitis, except that, in addition to the muco-purulent contents of the bronchi, fibrinous casts are also found.

Symptoms and Signs.—The *symptoms* do not differ essentially from those of simple bronchitis, except that there are paroxysms of severe dyspnœa from the plugging of a tube or of the tubes

with a fibrinous cast. The signs also are similar to those of ordinary bronchitis, unless a large tube is plugged, when there is an absence of all respiratory murmur over the lung to which the plugged tube leads.

Diagnosis.—The *diagnosis* is based on the presence of fibrinous casts in the sputa.

Prognosis.—The *prognosis* should be guarded, as about one-fourth of the cases terminate fatally.

Treatment.—The inhalation of lime water or of a solution of bicarbonate of soda to give relief by dissolving the fibrinous casts. Iodide of potassium has also been employed.

BRONCHIECTASIS.

Definition.—A dilatation of the bronchial tubes.

Causes.—1. *Inflammation*, and *softening* of the tubes, which makes them yield more readily to internal pressure, from secretions or inspired air.

2. *Accumulation* of *secretions*, which causes pressure in the tubes, besides softening them.

3. *Fibroid phthisis*, in which a contraction of the connective tissue occurs and thus pulls the walls of the tubes apart so as to cause dilatation.

Morbid Anatomy.—1. *Seat*. The usual seat is in the lower lobes and there are usually several dilatations.

2. *Forms*. The dilatations may be cylindrical or sacculated; frequently there are ridges projecting into the lumen of the dilatations.

3. *Changes in the mucous membrane and walls of the brouchi*. The mucous membrane frequently undergoes a change; the ciliated epithelium disappears and its place is taken by a flatter form.

There is an *increase* in the *connective* tissue surrounding the bronchial tubes, because the white blood cells which pass out during the inflammation are converted into connective tissue.

Symptoms.—1. *Respiratory*. *Cough* is a very prominent symptom in most cases; it is especially troublesome, as a rule, soon after getting up in the morning, because the secretions have accumulated during sleep and irritate the mucous membrane.

The *secretion* is muco-purulent in character, is usually very abundant, and is often fetid because it stagnates in the tubes and germs get in and set up decomposition.

2. *General*. In cases of old standing there is often considerable emaciation, due in part to the excessive discharge of pus and in part to the absorption of septic matters and the consequent fever.

Physical Signs.—1. On *inspection* and *palpation* there are no changes.

2. On *percussion* there is increased resonance, provided the dilatation is of some size, but as a rule it is not marked, and there may even be dullness because of the increased amount of connective tissue in the surrounding lung.

3. On *auscultation* large mucous râles or gurgling are heard unless the tube has just been emptied by coughing. If the surrounding lung tissue is more dense than normal, broncho-vesicular or broncho-cavernous breathing are heard.

Diagnosis.—It is distinguished from phthisis by (1) the absence, as a rule, of fever and (2) by the absence of the bacilli of tuberculosis from the sputa.

Prognosis.—The *prognosis* as to recovery is bad, but persons may live for years in comparative comfort.

Treatment.—The *treatment* is much the same as that of chronic bronchitis. Creosote, eucalyptol and turpentine are of service, and if the discharge is fetid, inhalations of carbolic acid or creosote are indicated.

BRONCHIAL ASTHMA.

Definition.—A disease characterized clinically by difficulty of breathing, especially on expiration, and by the presence of râles varying in size and character, and heard with great distinctness over both lungs. The disease occurs in paroxysms.

Pathology.—Two views are held as to the nature of the disease—1. The probable cause of the difficulty of breathing is a *spasm of the muscular coat* of the bronchial tubes.

2. Sir Andrew Clarke and others think the difficulty of treating is due to an *eruption*, like that of nettle rash, on the mucous membrane of the tubes, and the consequent obstruction to the ingress and egress of air.

Causes.—*Heredity* is a *predisposing* cause; not simply the occurrence of *asthma* in some ancestor, but the existence of *any* functional nervous disease. The existing causes are—

1. *Certain irritants*, differing in different people.
2. *Diseased condition of the nose, stomach, bowels, &c.*, which cause asthma by reflex action.
3. Certain diseases of the *lungs* or *heart*, such as emphysema, bronchial catarrh, mitral obstruction, &c.
4. *Retention of urinary matter* in the blood, which probably acts by irritating the respiratory centre in the fourth ventricle.
5. *Tumors of the fourth ventricle.*
6. *Irritation of the vagus* in its course.

Symptoms.—Sometimes *premonitory* symptoms occur, such as the passage of large quantities of limpid urine, but they are often absent.

1. The *onset* is usually rather sudden.
2. The *position* and *appearance* is quite characteristic. The patient is unable to lie down, and in severe cases leans forward. The face is more or less livid from interference with respiration, and the skin is bathed in sweat.
3. *Respiratory.* *Dyspnoea* is the most prominent symptom and this is especially great on *expiration*. *Cough* is usually very troublesome, but the *secretion* is tough and scant. *Speech* is embarrassed from the difficulty of respiration.
4. *Circulatory.* The pulse is small and weak, and the veins are distended in consequence of the interference with the exchange of gases in the lungs.

Physical Signs.—1. On *inspection* the chest is observed to be distended, and the respiratory movement slow and labored.

2. On *palpation* the rhoncal fremitus is very marked.
3. On *percussion* there is tympanitic resonance, in consequence of the over-distension of the air cells, because the *inspiratory* muscles are much stronger than the *expiratory*.
4. On *auscultation*, dry râles of all sizes and characters are heard in consequence of the narrowing of the tubes.

Diagnosis.—It is distinguished from—1. *Spasmodic affections* of the larynx by (1) the absence of râles, and (2) the greater difficulty in *inspiration* in the latter.

2. *Capillary bronchitis* by (1) the existence of fever, (2) the finer character of the râles, and (3) the greater difficulty in *inspiration* in the latter.
3. *Pulmonary oedema* and *congestion*, by the *mucoous* râles and liquid sputa in these affections.
4. *Cardiac dyspnoea* by the evidences of heart disease.

Prognosis.—The *prognosis* in simple asthma is always good as to life, but bad as to permanent recovery.

Complications.—*Emphysema* of the lungs and *bronchitis* are common complications.

Treatment.—1. To *remove the cause*, by relieving diseased conditions of the nose, stomach or bowels.

2. To *relax spasm*, by (1) *depressing* remedies, such as ipecac, lobelia and tobacco, or (2) by *sedatives* and antispasmodic, such as amyl nitrite, opium, chloroform, chloral, quebracho, nitre, grindelia, robusta, &c., or (3) by *stimulants*, such as alcohol and coffee.

3. To *build up the nervous system* by quinine, &c., and to lessen the tendency to spasm of the involuntary muscular fibre by nitroglycerine and other drugs of its class.

4. Certain drugs, such as *iodide of potassium*, have been found very useful in preventing the paroxysms, though their mode of action is not known. Probably the iodides act by allaying the inflammation and swelling of the bronchial glands.

5. Inhalations of the fumes from *nitre paper* are very useful in relieving the paroxysms.

Pyridin has also been of service.

PULMONARY EMPHYSEMA.

Definition.—A disease characterized by dilatation of the air vesicles with thinning and subsequent rupture of the partitions between them and destruction of many of the pulmonary capillaries.

Varieties.—The definition applies to vesicular emphysema; inter-lobular emphysema is the escape of air into the connective tissue between the pulmonary lobules.

Causes.—1. *Forced expiration* with the glottis closed, as occurs in violent straining, or among persons who use wind instruments.

2. *Impaired elasticity* and *nutrition* of the lungs; hence it occurs most commonly in elderly persons and is often hereditary.

3. *Solidification* or *collapse* of one part of a lung, or of one lung, may occasion emphysema of some other part, (compensatory emphysema)

Morbid Anatomy.—1. *Size.* The lungs are greatly distended and overlap the heart to a greater or less extent. The chest is enlarged and barrel-shaped.

2. *Changes in the air vesicles.* The air vesicles are distended and the partitions atrophied and ruptured so that large air spaces or sacs are formed, especially in the apices of the lungs where they are not protected by rigid chest walls. The capillary blood vessels on the walls of the alveoli undergo atrophy.

3. *Changes in neighboring viscera.* The permanent distension of the lungs with air forces the heart downwards and also causes depression of the liver and other abdominal viscera. In old cases there is hypertrophy and dilatation of the right side of the heart from obstruction to the flow of blood through the lungs.

Complications and Results.—1. *Bronchitis* is present as a complication in nearly every case of emphysema and adds to the gravity of the affection.

2. *Hypertrophy and dilatation of the right side of the heart* results from the destruction of the pulmonary capillaries and the consequent obstruction to the flow of blood through the lungs.

Symptoms.—1. *Respiratory.* *Dyspnoea*, increased by exertion, is the most prominent respiratory symptom. It is due to (1) the loss

of elasticity of the lung tissue and the consequent inability to empty the lungs, (2) to the destruction of the pulmonary capillaries, (3) to the loss of tone of the diaphragm and (4) to the accompanying bronchitis.

Cough and *expectoration* of tough mucus are commonly present and are due to the accompanying bronchitis.

2. *Circulatory*. The *pulse* is small and weak (unless arteriosclerosis is present as a complication), because but little blood passes through the lungs to the left side of the heart. The *venous system* is engorged in consequence of the dilatation of the right side of the heart, and the liver and spleen, are usually enlarged.

3. *Urinary*. The urine is scant and high-colored, from the lowering of the tension in the arteries.

4. *General*. In cases of long standing there is serious impairment of the flesh and strength in consequence of the defective circulation and nutrition.

Physical Signs.—1. On *inspection* the chest is observed to be barrel-shaped, from the inability to expel the air from the lungs, and the consequent distension; and in breathing the chest moves as a whole.

In severe cases pulsation of the jugulars occurs from tricuspid insufficiency.

2. The sound on *percussion* is vesiculo-tympanic, in consequence of the increased quantity of air in the chest.

3. On *auscultation* the respiratory murmur is found to be fainter than natural, because the amount of air retained in the lungs from the loss of elasticity interferes with the entrance of air.

Diagnosis.—It is distinguished from pneumothorax by the involvement of both lungs and its gradual onset.

Prognosis.—The disease is dangerous only from the complications and results, which are always serious.

Treatment.—1. To *improve nutrition* by tonics, especially cod-liver oil.

2. To *prevent or lessen complications* by residence in a dry climate and by the proper treatment of bronchitis, &c.

3. To *lessen expansion* of the *alveoli* by *exhaling* into a rarified atmosphere and by inhaling compressed air. Compression of the chest by mechanical means, during expiration, for a short time every day is of great service.

4. To *relieve dyspnoea* by *quebracho* and *grindelia robusta*.

ACUTE LOBAR PNEUMONIA.

Synonyms.—Croupous pneumonia, pneumonic fever, lung fever.

Definition.—An acute, infectious disease, characterized by its sudden onset, high temperature and by inflammation of one or more lobes of the lungs.

Causes.—1. A *germ* is probably the essential cause of pneumonia; but it is doubtful whether the oval coccus described by Friedlander is always the infecting agent.

2. *Age.* The disease may occur at any age, but is most common in young adults.

3. *Sex.* It is rather more common in men than in women, because they are more exposed to the existing causes, such as change of temperature.

4. *Depression of vital powers* from any cause, such as alcoholism, malaria or any severe illness, which renders the system less resistant than natural. Organic heart diseases probably act in the same way.

5. *One attack renders a person more liable*, apparently, to subsequent ones, partly, perhaps, by impairing the general health and partly because of some natural want of resistance to the germs in some people.

6. A *changeable climate* and the winter and spring seasons undoubtedly exert a causative influence, chiefly, perhaps, by causing contraction of the cutaneous blood vessels and consequent collateral hyperæmia of internal organs.

Morbid Anatomy.—The *lower lobe* of the *right lung* is most commonly involved.

The disease presents three stages.—*First stage, or stage of engorgement*—1. The *color* is darker than natural, because the amount of blood in the lungs is increased.

2. In *consistence* the lung is softer than natural and pits on pressure because of the increased amount of fluid in it (liquid exudate).

3. *On section* the air cells are found to contain a more or less bloody and viscid fluid; the capillaries are distended with blood and the epithelium lining the alveoli has undergone fatty degeneration and in some places is desquamating.

Second stage or stage of red hepatization.—1. The *color* is very dark.

2. The *consistence* is very firm because the alveoli are filled with a fibrinous exudate.

3. *On section* or fracture the alveoli are found to be filled with fibrinous plugs, giving the lungs a granular appearance. There is little or no *liquid* discharge on section in this stage.

Third stage or stage of gray hepatization.—1. The *color* is gray and mottled—redder in some parts than in others.

2. The *consistence* is softer than in the second stage.

3. *On section*, the alveoli are found to be filled with white blood (or pus) cells, red blood corpuscles and granular matter, the white cells being in excess.

It is doubtful whether this stage ever occurs except in fatal cases. It is sometimes called the stage of purulent infiltration.

The *duration* of the first stage is from six to twenty-four hours; that of the second stage from five to nine days.

Symptoms.—1. The *onset* is nearly always sudden.

2. The *nervous* symptoms are, (1) a *chill* at the commencement, which is usually violent in character, (2) *pain* in the chest, which is due to the accompanying pleurisy, (3) *headache* and subsequently delirium, due to the circulation of venous blood probably containing leucomaines.

3. The *temperature* rises rapidly to 104° , 105° , or possibly even higher, and continues at this point for from five to nine days, when it usually falls suddenly to the normal.

4. The *respiratory* symptoms are, (1) *rapid* and *panting* breathing, due to the lessening of respiratory space.

(2.) *Cough*; usually very troublesome, which is due to the irritation of the terminal filaments of the sensory nerves in the lungs, (3) *expectoration* at first of a tough mucus which soon becomes stained with blood (*rusty sputa*) and is exceedingly tenacious, and later becomes more liquid and copious from the liquidation and breaking down of the exudate.

5 *Circulatory*. The *pulse* is rapid and full in the early stages because of the high temperature, but later on it often becomes weak from the albuminoid degeneration of the heart muscle.

6. *Cutaneous*. The *skin* is very hot and dry in the early stages, but when the crisis comes there is often a profuse sweat and sometimes even before the crisis sweating occurs. The face has a dusky look and there is a flush in one cheek.

7. *Digestive*. *Anorexia* is the rule, and nausea and vomiting may occur; the bowels are usually constipated, but in very severe cases troublesome diarrhoea sometimes occurs.

8. *Urinary*. The *urine* is scant and high colored and the chlorides are greatly diminished in quantity. Albumin is frequently present in small amount, and there may be a considerable quantity, especially when the apex of the lung is involved. The albuminuria is probably caused by the elimination of leucomaines by the kidneys.

Physical Signs.—In the *first stage* or stage of *congestion*.—1. On *inspection* the respirations are seen to be hurried and the movements on the affected side are less free than normal, (1) because the air cannot enter the lung as well as in health, and (2) because of the pain occasioned by the position of the inflamed pleural surfaces against each other.

2. On *palpation* the vocal fremitus is slightly increased because of the commencing consolidation of the lung.

3. On *percussion* there is some dullness for the same reason.

4. On *auscultation* crepitant râles are heard over the inflamed lung: these râles are heard especially during inspiration, and are probably due to the pulling apart of the agglutinated walls of the air vesicles. *Pleuritic* friction sound is usually heard also because pleurisy is such a common complication of pneumonia.

In the *second stage* or *stage of consolidation*.—1. *Inspection* shows an absence of respiratory movement over the affected lung because it is impervious to air.

2. On *palpation* the vocal fremitus is greatly increased because vibrations are carried better through a solid than a gaseous medium.

3. On *percussion* there is very marked dullness because there is very little air in the lung.

4. On *auscultation* there is *bronchial breathing* because the solidified lung tissue conveys the sound from the bronchial tubes to the ear far better than the healthy lung, which contains air. The vesicular murmur is lost because the air vesicles are filled with a firm substance and no air can enter them.

In the *third stage*, if the lung tissue is clearing up, the signs are very much like those of the first stage.

If *purulent infiltration* occurs, *liquid* râles are heard over all parts of the diseased lung.

Diagnosis.—1. From *pulmonary congestion* and œdema, pneumonia is distinguished by the chill, sudden onset and high temperature in the latter.

2. From *acute capillary bronchitis* by the different onset and by the fact that bronchitis involves both sides and does *not* cause solidification of the lung with the consequent physical signs.

3. From *acute pleurisy* by the absence of dullness on percussion and bronchial breathing in pleurisy.

4. From *hypostatic congestion* by the sudden onset and high temperature of pneumonia, which is not seen in hypostatic congestion.

5. From *lobular pneumonia* by the fact that this disease is always secondary; that the lobules of the lungs are involved and that the fever is less high.

Complications.—1. *Pleurisy* is a very common complication from extension of the inflammation to the pleura overlying the diseased lung.

2. *Pericarditis* sometimes occurs, probably from extension.

3. *Meningitis* is a rare complication, and may occur in any acute infectious disease.

4. *Congestion* of the *liver* and *spleen* generally occurs, as in other infectious diseases.

5. *Gastro-intestinal catarrh* is sometimes a serious complication, but is not a very common one.

Nature of Pneumonia.—Pneumonia is probably an acute infectious disease, because it resembles them in the following particulars—1. Character of invasion.

2. Cyclical course—from four to nine days.

3. It is not caused by injuries.

4. It is sometimes epidemic and apparently contagious.

5. The complications are like those of the acute infectious diseases.

6. The general symptoms are often out of proportion to the local disease.

Terminations.—1. *Resolution* and absorption of the inflammatory exudate is much the most common termination.

2. *Purulent infiltration* is especially apt to occur in elderly and debilitated persons, and nearly always terminates fatally.

3. *Abscess* and *gangrene* are rare terminations.

4. *Chronic pneumonia* is also a rare termination.

Prognosis.—The *prognosis* in uncomplicated cases and in previously healthy persons is usually good; but is materially influenced by the following circumstances:—1. *Age*. The danger is very much greater in old persons than in vigorous adults for obvious reasons.

2. *Extent and situation* of the disease; pneumonia of the apex is more serious than pneumonia of the base, and of course the greater the surface involved the greater is the danger.

3. The *general condition* of the patient is of great importance in prognosis; debility from any cause greatly increases the danger. The circumstances, however, which most seriously influence the prognosis are *intemperance* and *malaria*.

4. A very *high temperature*, if prolonged, renders the prognosis unfavorable.

5. *Complications* of any kind increase the danger; especially is this true of *pericarditis* and *malarial poisoning*.

Causes of Death.—1. *Asphyxia* is sometimes a cause of death in double pneumonia.

2. *Asthenia*, from the prolonged high temperature and the degeneration of the heart muscle, is the most common cause.

3. *Thrombosis* in the cavities of the heart or of the pulmonary artery is an occasional cause of death.

4. *Urinæmic poisoning* occasionally occurs when kidney complications are present.

Treatment.—The indications and means of fulfilling them are briefly as follows—1. To *relieve pain* by morphia, cups and poultices.

2. To *lessen the amount of blood in the lungs*, by (1) remedies which dilatate the vessels in the skin, such as poultices externally, and aconite, veratrum, ipecac, &c., internally; (2) lessening the force and frequency of the heart's action by aconite, veratrum, &c.

3. To *reduce the temperature* by cold baths (which must be used with great caution), phenacetine, antipyrine, quinine, &c.

4. To *facilitate respiration*, especially in double pneumonia, by inhalations of oxygen, and possibly by bleeding in robust patients.

5. To *sustain strength*, by food and stimulants, and by digitalis when heart failure is threatening. Carbonate of ammonia is especially useful.

6. To *avoid complications* by proper attention to diet and other hygienic measures.

LOBULAR PNEUMONIA.

Synonyms.—Broncho-pneumonia, catarrhal pneumonia, secondary pneumonia.

Definition.—An inflammatory affection commencing in one or more lobules of the lung and frequently spreading to adjacent lobules.

Causes.—1. It is *secondary* to bronchitis or collapse of the lung.

2. It is more common in *children* than in adults, because the small size of their tubes renders them more liable to collapse.

3. *Bad hygienic* surroundings by causing debility increases the liability to it.

4. *Injuries* to the *lungs*, or the *inhalation* of noxious matters, as in typhoid fever, act as causes by direct irritation.

5. It is frequently secondary to, or a complication of, measles, whooping cough, diphtheria, etc.

Morbid Anatomy.—1. *Seat.* Usually in the lower back part of the lungs, because inhaled or inspired substances are most apt to lodge there.

2. *Extent.* The disease commences usually in one lobule or in several which are scattered through the lungs, but several such lobules may unite to form a large patch.

3. *Color.* The color is *dark* from the presence of blood in the vessels in greater quantity than normal.

4. The *consistence* is firmer than that of the healthy lung, but it is less resistant to a tearing force because of the saturation with the exudate.

5. The *bronchi* are inflamed and contain a muco-purulent fluid.

6. The *alveoli* contain the exudate characteristic of catarrhal inflammation, namely, pus cells, large cells much like epithelium,

a certain amount of granular detritus and mucus. The cells lining the alveoli undergo granular and fatty degeneration, and peel off. The capillaries are distended with blood.

Symptoms.—1. Those of the preceding disease.

2. *Respiratory.* The *cough* is usually troublesome, because of the accompanying bronchitis. The *expectoration* is thinner, more muco-purulent in character than in lobar pneumonia because of the catarrhal inflammation; it is usually blood-stained, but in children the sputa may be swallowed, so that the amount and character cannot be determined. The degree of *dyspnea* depends upon the amount of lung tissue involved and upon the degree of obstruction of the bronchial tubes. As a rule it is much less than in lobar pneumonia, because less lung tissue is implicated and the temperature is not so high.

3. *Circulatory.* The *pulse* is usually rapid and feeble, chiefly in consequence of the debilitating effect of the preceding disease.

4. *Digestive.* *Anorexia* is the rule, and nausea and vomiting occasionally occur. The bowels are usually constipated from the loss of tone of the muscular coat, but intestinal catarrh may occur.

5. *Nervous.* A *chill* of moderate intensity usually occurs at the commencement of the attack. A stitch in the side is also a frequent symptom, but it is less frequent and less severe than in lobar pneumonia.

6. *General.* *Debility* and *pallor* are usually very marked in cases of lobular pneumonia, but they are attributable rather to the preceding disease than to the pneumonia itself.

Physical Signs.—1. On *inspection* there is usually but little change from health in the fulness of the respiration, because the amount of lung tissue involved is not sufficient to interfere with the inflation of the lungs.

2. On *palpation*, if the hand be placed over the solidified lung tissue, the vocal fremitus will be found to be increased.

3. On *percussion*, there is dullness over the inflamed spot because of the diminution in the quantity of air in that part of the lung.

4. On *auscultation*, there is bronchial breathing for the same reason, and there are, as a rule, râles varying in size, but chiefly sub-crepitant, which are due in great part to the presence of mucus in the tubes.

Diagnosis.—It is diagnosed from—1. *Lobar pneumonia*, by the fact that it is secondary, that its onset is more gradual, that a much smaller surface is involved, and that the temperature is not so high and the disease does not run a definite course, as lobar pneumonia does.

2. *Capillary bronchitis*, by the rusty sputa and bronchial breathing, which are absent in bronchitis.

3. *Acute tuberculosis*, by the lower temperature, the slighter degree of prostration, and the absence from the sputa of the bacilli of tuberculosis.

4. *Collapse of the lung*, by the sudden occurrence of the latter and by the absence of bloody sputa and fever.

Complications.—1. *Capillary bronchitis* is a very common complication, and is very often a cause of the pneumonia.

2. *Fibroid induration* of the lung in cases of long duration.

3. *Tuberculosis* is a not uncommon complication, because the lobular pneumonia furnishes a suitable soil for the development of the bacilli of tuberculosis.

4. *Pleurisy* from extension of the disease to the overlying pleura.

5. *Acute intestinal catarrh*, which is especially common in children, and is due, in part, at least, to the swallowing of substances which should be expectorated.

Prognosis.—The *prognosis* is usually more serious than that of lobar pneumonia, because of the debility from the preceding disease. The *duration* is always, as a rule, longer than that of lobar pneumonia.

The *circumstances influencing the prognosis* are, (1) the preceding disease, (2) the age and health of the patient, (3) the height of the fever.

Terminations.—The terminations are—1. *Resolution*, which is the most frequent termination in healthy persons.

2. *Chronic pneumonia*, which is especially apt to occur in debilitated persons surrounded by bad hygienic conditions.

3. *Pulmonary phthisis*, from the caseation and softening of the inflammatory exudate.

4. *Bronchiectasis*, from the softening of the tubes and from the fibroid induration of the lung tissue, which occurs in cases of old standing.

Causes of Death.—1. *Asphyxia*, when a number of lobules are involved, or when capillary bronchitis co-exists.

2. *Asthenia*, or exhaustion, which is much the most common cause of death.

3. *Complications*, especially capillary bronchitis.

Treatment.—1. To cure the preceding disease.

2. To draw blood to the skin by hot applications, such as poultices, turpentine stupes, &c.

3. To lessen fever by phenacetine, quinine, &c.

4. To sustain strength by stimulants and food and by carbonate of ammonia and digitalis, if the heart's action is weak.

5. To avoid hypostasis by frequent changes of position and by the use of cardiac stimulants.

6. To *prevent evil results*, such as chronic pneumonia, by stimulants, tonics, fresh air and nourishing food.

HYPERÆMIA OF THE LUNGS.

Definition.—An excessive quantity of blood in the lungs without actual inflammation.

Varieties.—1. *Active*, due to the excessive influx of *arterial* blood.

2. *Passive*, due to the obstruction to the outflow of *venous* blood.

Causes.—The causes of *active* hyperæmia of the lungs, are:—

1. *Alcohol*, which increases the force and frequency of the heart's action and dilates the blood vessels.

2. *Exertion*, which acts in a similar manner.

The cause of *passive* hyperæmia of the lungs is usually *obstruction* at the mitral orifice, but *regurgitation*, or leaking at the same orifice, will have a similar effect.

Morbid Anatomy.—1. The *color* of the lungs is darker than normal from the excessive amount of blood in them.

2. The *consistence* of the lungs is increased because the relative amount of air is lessened.

3. The *capillary vessels* are distended with blood.

4. The *cells* of the *alveoli* show albuminoid degeneration.

5. In cases of long standing, *brown induration* occurs from the deposit of blood pigment in the tissues; this occurs especially in passive hyperæmia.

Symptoms.—1. *Respiratory.* *Cough* is usually quite troublesome. The *expectoration* in severe cases of active hyperæmia is blood stained; in the passive form it is usually quite copious and liquid. *Dyspnoea* is present in greater or less degree from a simple sensation of tightness across the chest to decided difficulty of breathing. It is due to the over-filling of the lungs with blood and the consequent failure in the proper exchange of gases.

2. *Circulatory.* In the *active* form the pulse is full and strong; in the *passive*, usually weak and feeble, the pulmonary second sound accentuated from the increased pressure in the pulmonary artery, and there is engorgement of the systemic veins in cases of long standing.

3. The *temperature* is not elevated.

Physical Signs.—1. On *percussion* there is usually slight dullness because the air space is lessened.

2. On *auscultation* there is sometimes broncho-vesicular breath-

ing and râles are heard. In the passive form they are usually abundant and moist.

Diagnosis.—The *diagnosis* is based on:—1. The *dyspnœ*.

2. The *bloody sputa*.

3. The *absence* of fever.

4. The existence of *causative conditions*.

Prognosis.—In the *active* form the prognosis is usually good, because the causes can be removed.

In the *passive* form it is usually bad, because the causes are incapable of removal.

Treatment.—To lessen the amount of blood in the lungs.—1. In the *active* form by (1) remedies which lessen the amount of blood, such as bleeding or wet cups, (2) dilating the vessels of the skin by poultices, &c., (3) lessening the force and frequency of the heart's action by aconite, &c.

2. In the *passive* form by agents which *increase the force of the heart*, such as stimulants and digitalis.

To *relieve the pulmonary œdema*, which often occurs from passive hyperæmia, hydragogue cathartics are indicated.

ŒDEMA OF THE LUNGS.

Definition.—The accumulation of fluid in the air vesicles and interstitial tissue of the lungs.

Causes.—Pulmonary œdema is always *secondary* to—

1. *Bright's disease*.

2. *Passive hyperæmia*, which causes overfilling of the veins and consequent transudation of serum from malnutrition of the walls of the vessels.

3. *Certain exhausting diseases* which act by inducing malnutrition of the walls of the vessels, and also weakness of the heart.

4. *Old age* and *position* (hypostasis) probably act in the same way.

Morbid Anatomy.—The *morbid anatomy* consists solely in the presence of an excessive amount of watery fluid in the air cells and interstitial tissue of the lungs.

Symptoms.—1. *Respiratory*. *Dyspnœa* is usually excessively severe from the loss of ærating space. *Cough* is troublesome until the patient becomes benumbed by the carbonic acid which is retained in the blood. The *expectoration* is liquid in character, but may be viscid.

2. *Circulatory*. The *pulse* is usually very feeble and the skin cyanotic.

Physical Signs.—1. On *percussion* there is more or less dullness in consequence of the presence of fluid in the air cells.

2. On *auscultation* great numbers of liquid râles, varying in size, are heard over both lungs.

Diagnosis.—It is distinguished from—1. *Pneumonia* by the absence of fever, the absence of bronchial respiration, as a rule, and by the liquid character of the sputa.

2. *Hydrothorax* by the liquid râles heard on auscultation, and by the absence of flatness on percussion.

3. *Capillary bronchitis* by the different causative diseases in the two cases and by the fever which is present in bronchitis.

Prognosis.—The *prognosis* is usually bad because of the gravity of the causative disease.

Treatment.—The indications of treatment and the means of fulfilling them are—1. To *increase* the *power* of the *heart* by stimulants and digitalis.

2. To *draw blood* to the *surface* by dry cups; poultices are not proper here, because they may cause debility.

3. To *remove the water* by diaphoretics, diuretics and hydragogues.

4. To *lessen dyspnœa* by the inhalation of oxygen.

INFARCTION OF THE LUNGS.

Definition.—The extravasation of blood into the substance of the lungs and air vesicles from the plugging of a branch of the pulmonary artery. (See Infarction in general pathology.)

Causes.—1. *Disease of the heart*, especially of the mitral valve, leading to stagnation in the right side of the heart and consequent coagulation of the blood there—a piece of such coagulum being subsequently swept into the pulmonary artery.

2. The lodging in the pulmonary artery or its branches of an embolus from the systemic veins.

Morbid Anatomy.—1. The *seat* of an infarction is usually near the surface of the lung.

2. It is triangular in *shape* from the arrangement of the vessels; it varies in size; it is firm in consistence, but tears readily.

3. The *color* is dark from the great increase in the amount of blood in that part of the lung.

4. The *pleura* over the infarction is inflamed.

5. The *blood vessels* and alveoli are filled with venous blood.

Symptoms.—The symptoms and signs depend in great measure on the size of the infarction.

1. The *respiratory* are cough, dyspnœa, tightness across the chest and the expectoration of dark blood. The cause of these symptoms is the engorgement of the part of the lung involved and the consequent diminution of breathing space.

2. The *circulatory* symptoms are engorgement of the venous system and usually weak and rapid pulse.

3. The most prominent symptom connected with the nervous system is pain, which is due to the accompanying pleurisy.

4. The *temperature* is slightly elevated, but rarely rises above 102°.

Physical Signs.—1. On *percussion* there is dullness, the extent of which depends, of course, upon the size of the infarction.

2. On *auscultation* there are often coarse mucous râles, and bronchial breathing is also present after a short time.

Diagnosis.—The *diagnosis* is based on (1) the presence of diseases which would lead to infarction and (2) the character of the sputa.

Termination.—1. *Resolution*, if the infarction is not large and the general health is good.

2. A *cyst* may form from inflammation around the infarction and the partial absorption of the infarction itself, so that the contents of the cyst are cheesy or chalky matter.

3. *Pneumonia* may also occur as a termination and is usually of a very severe grade.

4. *Gangrene* or *abscess* may occur when the embolus leading to the infarction is infective.

Prognosis.—The *prognosis* depends upon the cause and nature of the embolus, but is always serious except in the mildest cases.

Treatment.—The *treatment* consists in sustaining strength by rest, nutritious food and stimulants and the stimulation of the heart by digitalis.

GANGRENE OF THE LUNG.

Varieties.—1. Diffuse.

2. Circumscribed.

Causes.—1. The *entrance* into the lungs of organic matters, such as particles of food, pieces of tissue from cancerous growth, &c. This is especially apt to occur in insane patients and drunkards, or in persons who have paralysis of the muscles of deglutition.

2. *Certain pulmonary affections*, especially interstitial pneumonia may cause it.

Morbid Anatomy.—1. In the *diffused* form, the whole lung may be softened and gangrenous without any line of demarkation.

2. In the *circumscribed* form the gangrenous patch is usually situated in the lower back part of the lung, and is surrounded by a form of catarrhal pneumonia.

Symptoms.—1. *Respiratory.* Fetid breath, more or less dyspnoea and cough, and the expectoration of shreds of lung tissue, constitute the respiratory symptoms and need no explanation.

2. The *general symptoms* are those of exhaustion.

3. The *temperature* is usually elevated, 103° to 105° , but if there is a free vent for the gangrenous matter there may be no fever.

Complications.—Empyema, intestinal disturbances and abscess of the brain may occur as complications.

Diagnosis.—The *diagnosis* is based on (1) the fetor, and (2) the presence in the sputa of shreds of lung tissue. Probably the withdrawal of fetid gases from the pleural cavity by aspiration would be useful.

Prognosis.—The *prognosis* is bad, but in circumscribed cases recovery may occur.

Treatment.—The indications are—1. To *sustain strength* by tonics, nutriment and stimulants.

2. To *lessen the fetor* of the discharge by inhalations of turpentine and the use of turpentine internally. Carbolic acid and creosote may be used in the same way.

3. *Thoracotomy* and the removal of the gangrenous tissue is sometimes useful.

COLLAPSE OF THE LUNG.

Definition and Synonym.—In collapse of the lung or atelectasis, the pulmonary tissue in a part of the lung is devoid of air.

Causes.—1. *Age.* It is much more common in childhood than in after life, because the exciting causes, such as whooping cough, &c., are more common then.

2. *Debility.* Weakly children are more liable to it than strong ones, because the respiratory muscles do not act with sufficient force to draw air into the lungs.

3. *Bronchitis* is one of the commonest of the existing causes. A tube becomes plugged with mucus and the air in the corresponding part of the lung is gradually absorbed.

4. *Whooping cough* is also a common cause; the air is forced out of a part of the lung during violent expiration and the lung is not re-inflated.

5. *Intra-thoracic pressure*, as from pleural effusion, is also a cause.

Morbid Anatomy.—1. *Seat*. The usual seat of atelectasis is on the surface of the lower back part of the lungs.

2. *Color and consistence*. The color is bluish and the consistence firmer and tougher than natural. The lung is airless and does not crepitate. It sinks in water.

3. The *bronchi* usually contain mucus and are occluded by it.

Symptoms.—1. *Respiratory*. The respirations are feeble and shallow. Cough is usually present because of the accompanying bronchitis.

2. *Circulatory*. The pulse is rapid and feeble, and there is blueness of the surface because of the obstruction to the flow of blood through the lungs.

Physical Signs.—1. On *inspection* it will be seen that the movements of the chest are not as free as they should be, and in bad cases there is sinking-in of the supra-clavicular region and the abdomen on inspiration, in consequence of the failure of inflation of the lung.

2. On *percussion* there is dullness over the collapsed spots; and

3. On *auscultation* bronchial breathing is heard over them.

Diagnosis.—It is diagnosed from—1. *Pneumonia*, by the absence of fever and bloody sputa.

2. *Tuberculosis*, by the absence of fever and the seat of the disease—at the lower part of the lung.

Pleurisy, with *effusion*, by the absence of flatness on percussion, and the change of level of the fluid in pleurisy, when the position is changed.

Results.—A very common result of collapse of the lung is *broncho-pneumonia*.

Prognosis.—The *prognosis* depends, in great measure, on the previous state of health, but it is usually bad, especially after whooping cough.

Treatment.—1. To *stimulate respiration*, by cold douches, strychnine and other respiratory stimulants.

2. To *relieve the causative conditions*, such as bronchitis.

3. To *improve the general health*, by nutritious food, cod liver oil and stimulants.

ACUTE PLEURISY.

Definition.—An acute inflammation of the pleura.

Causes.—1. *Exposure* to cold and dampness is probably a cause of acute pleurisy.

2. *Injuries*, such as fracture of the ribs or wounds of the chest, will cause it.

It is often an accompaniment of *other diseases*, such as pneumonia, Bright's disease and the acute infectious diseases.

Morbid Anatomy.—1. The *color* of the membrane is changed so that it becomes reddened and cloudy.

2. In *texture* it is softened and the cells undergo granular degeneration.

3. The *exudate* is fibrinous in character, but there is probably in all cases more or less liquid exudate also.

The pleural surfaces are roughened by the exudate.

Results.—As a result of the inflammation the pleura becomes *thickened* and *adhesions* are formed between the costal and pulmonary pleura.

Symptoms.—1. *Nervous*. Pain is one of the most striking of the symptoms of acute pleurisy; it is due to the rubbing against each other of the inflamed surfaces, and is increased by breathing.

2. *Respiratory*. The respiration is hurried and shallow because of the pain which it occasions. There is more or less *cough*, but no expectoration in simple cases.

3. *Circulatory*. The *pulse* is small and rapid.

4. The *temperature* is elevated but rarely goes above 102°.

Physical Signs.—1. On *inspection*, the respiratory movements are seen to be shallow and hurried.

2. On *palpation*, friction fremitus can often be detected, caused by the rubbing against each other of the surfaces roughened by the exudate.

3. On *percussion*, there is no change unless the amount of effusion is large, when there will be flatness over the seat of effusion.

4. On *auscultation*, a friction sound is heard in consequence of the rubbing against each other of the inflamed surfaces. If there is much liquid exudate the surfaces are held apart and the friction sound is absent.

Diagnosis.—It is diagnosed from—1. *Pneumonia*, by the absence of dullness on percussion and of rusty sputa, and also by the temperature, which is much higher, as a rule, in pneumonia.

2. *Intercostal neuralgia*, by the absence of fever, as a rule, in neuralgia and the absence of the physical signs of pleurisy.

Prognosis.—The *prognosis* of acute pleurisy is usually good, but adhesions always result.

Treatment.—1. To *relieve pain* by opium and rest, and by counter-irritants, such as turpentine stupes.

2. To *build up the general health* after the acute stage of the attack has passed, by tonics, such as iron and cod-liver oil.

3. To *promote the free expansion of the lungs* after the acute stage has passed, by means of gymnastic exercises.

SUB-ACUTE OR SEROUS PLEURISY.

Definition.—*Frequency.* A sub-acute inflammation of the pleura with serous exudate usually in considerable amount. It is quite frequent.

Causes.—1. The disease is usually *secondary* to Bright's disease or phthisis.

2. *Exhaustion* or *exposure* to cold and dampness may cause it, however; they usually act as exciting causes.

Morbid Anatomy.—1. One side only is usually involved.

2. *Color* and *consistence.* The pleura loses its lustre and becomes softened.

3. The *exudate* is liquid and may nearly fill one side of the chest.

4. The *lung* on the affected side is collapsed and pressed upward and backwards. The heart is pushed toward the sound side by the effusion.

5. At a late stage after the fluid is removed there are *adhesions* and the chest may be retracted if the lung does not expand again.

Symptoms.—1. *Respiratory.* The dyspnœa is usually considerable from diminution of respiratory space which is caused by the pressure of the effusion. It is greatly increased by exertion.

2. *Circulatory.* The pulse is rapid and feeble, especially on exertion.

3. The *temperature* is rarely elevated unless the pleurisy is associated with phthisis.

4. The *general symptoms* are pallor and exhaustion.

Physical Signs.—1. On *inspection* the chest is enlarged on the diseased side, and if the effusion is great there is bulging of the intercostal spaces. Respiratory movement is absent.

2. On *palpation* there is loss of vocal fremitus.

3. On *percussion* there is flatness over the effusion.

4. On *auscultation* there is absence of all respiratory sound over the effusion and bronchial respiration over the upper back part of the chest where the compressed lung is situated.

Diagnosis.—1. From *pneumonia* and *phthisical* consolidation, pleurisy is distinguished by the absence of bronchial breathing over the effusion and the absence of fever, as a rule.

2. From a *tumor* in the chest it is diagnosed by the result of aspiration.

Prognosis.—The *prognosis* is usually favorable, but *phthisis* is a probable *sequel*.

Treatment — 1. The first indication is to *improve* the *general health* by means of the syrup of iodide of iron, nutritious food, stimulants and fresh air.

2. To *remove the effusion* when the respiration is seriously embarrassed by means of concentrated saline solutions and aspiration.

EMPHYEMA.

Definition and Varieties.—A collection of pus in the pleural cavity; it may be *primary* or *secondary* to some injury, or to phthisical perforation of the pleural cavity.

Causes.—1. An *injury* which admits germs to the pleural cavity.

2. *Phthisis* which perforates the pleural cavity and allows germs to enter.

3. *Debility* which renders the tissues less able to resist the attacks of the germs of suppuration.

Morbid Anatomy.—1. The *seat* is nearly always on one side; the *extent* is variable; sometimes there are several collections of pus, surrounded and kept in place by adhesions.

2. The *exudate* is purulent and may be very large in quantity.

The *condition* of the *lung* is the same as in pleurisy with serous exudate.

Symptoms.—The *symptoms* are those of serous pleurisy, and, in addition, the evidences of pus formation, *high temperature*, *sweats*, *exhaustion* and *emaciation*.

Physical Signs.—The *physical signs* are precisely similar to those of serous pleurisy. Sometimes an external opening forms and a fistula results.

Diagnosis.—The *diagnosis* can only be made with certainty by aspiration.

Prognosis.—The *prognosis* is usually bad without treatment, but favorable with proper management.

Results — 1. *Perforation*, and discharge of pus—

- (1) through thoracic walls;
- (2) through abdominal walls;
- (3) into a bronchus;

(4) into the abdominal cavity, colon or stomach ; rarely into the pelvis of the kidney.

2. *Necrosis* of one or more *ribs*.

3. *Cheesy or calcareous* degeneration of the exudate.

4. *Collapse* of the *chest wall* after the discharge, with recovery.

Treatment.—1. To *improve* the *general* health and sustain strength by nourishing food and stimulants.

2. *Aspiration* and withdrawal of pus may be tried in the case of children.

3. *Free opening* in the scapular line, with thorough and continuous drainage.

4. *Washing* out the chest with a mixture of compound tincture of iodine and water (one part to four).

5. In old cases the *excision* of a part of the *chest wall* so as to allow the chest wall and lung to approach each other and heal more readily.

PNEUMOTHORAX.

Definition and Synonyms.—An accumulation of air in the pleural cavity.

It is also called *pyo-pneumothorax* and *hydro-pneumothorax*, because the entrance of air with the germs in it sets up inflammation and leads to the formation of an exudate, serous or purulent in character.

Causes.—1. *Phthisis* is the most common cause. A cavity opens into the pleural cavity and allows the entrance of air.

2. *An injury*, which causes an opening into the pleural cavity.

3. *Emphysema*, the entrance of air being due to the bursting of an enlarged air vesicle.

4. *Empyema*, which bursts into a bronchus.

Morbid Anatomy.—1. The *position* of the lung is changed, so that it occupies the upper back part of the thorax, and is collapsed.

2. The *changes in the pleura* are those of inflammation.

Symptoms.—1. Sudden and extreme *dyspnea* from the collapse of the lung.

2. *Collapse* from the general shock.

3. If life is prolonged, *fever*, *exhaustion* and dropsy occur from pus formation.

Physical Signs.—1. On *inspection*, the chest is found to be

enlarged on the affected side, and the respiratory movements are absent.

2. On *palpation* vocal fremitus is absent.

On *percussion* there is tympanitic resonance over that part of the chest containing an excess of air and flatness over the lower part where the fluid is.

4. On *auscultation* there is an absence of all respiratory sound except at the upper back part of the chest where there is bronchial breathing over the collapsed lung; succussion causes metallic tinkling.

Diagnosis.—It is distinguished from—1. *Empyema*, by the sudden occurrence and the tympanitic resonance above the level of the fluid.

2. A *phthisical cavity*, by the cavernous respiration and the gradual formation of a pulmonary cavity.

Prognosis.—The *prognosis* is almost always unfavorable. Recovery may occur from closure of the opening.

Treatment.—The *treatment* consists in relieving pain by morphia, quebracho and hot applications to the chest.

HYDROTHORAX.

Definition.—A collection of fluid in the thoracic cavity not due to inflammation.

Causes.—1. *Venous hyperæmia* from any cause especially disease of the mitral valve.

2. Bright's disease.

Symptoms.—1. The *appearance* of the patient is striking; there is pallor and cyanosis and it is difficult or impossible for him to lie down, because the abdominal viscera will push against the diaphragm and still farther hinder the action of the heart and lungs.

2. *Dyspnœa* occurs in greater or less degree from the pressure on the lungs.

3. The *pulse* is small and rapid from the pressure upon the heart and vessels which prevents them from being properly filled.

Physical Signs.—*Absence* of *respiratory movement* and of vocal resonance, flatness on percussion and absence of respiratory sound over the lower part of the chest where the fluid is.

Diagnosis.—It is distinguished from—1. *Pleurisy* with *serous effusion* by the fact that pleurisy occurs only on *one* side and hydrothorax on both.

2. *Pulmonary œdema* by the liquid râles in œdema and the absence of respiratory sound in hydrothorax.

Prognosis.—The *prognosis* depends on the nature of the previous disease. The fluid can usually be readily removed, but so long as the cause persists is liable to return.

Treatment.—1. To *remove* the *fluid* by (1) hydragogue cathartics, such as the saline purgatives, elaterium, &c ;

(2) *diuretics* and *diaphoretics* ;

(3) *aspiration* which is certain and very little dangerous.

2. To *improve* the *general health* by tonics, &c.

PULMONARY PHTHISIS.

(Phthisis—Pulmonary Consumption—Pulmonary Tuberculosis.)

Definition.—An inflammatory affection of the lungs, caused by the *bacillus tuberculosis*, and characterized by a great tendency to caseation and softening, or by the formation of connective tissue. The course is chronic and the disease one of the most frequent known.

Causes.—1. The *bacillus tuberculosis* is the essential cause. (See Tubercle and Tuberculosis for description and favorable conditions for the development of this germ.)

2. A certain *predisposition*, which may be (1) hereditary, (2) acquired. Nothing is known of the nature of the *hereditary* predisposition.

The *acquired* predisposition is brought about by (a) bad hygienic conditions, especially dampness and over-crowding ; (b) bronchitis and catarrhal pneumonia ; (c) pleurisy ; (d) pregnancy and lactation ; (e) diabetes ; (f) typhoid fever ; (g) measles.

Most of these predisposing causes act by lowering the vitality of the tissues ; pleurisy acts by lessening the freedom of the chest movements, and thus permitting the germs to remain in contact with the bronchioles or alveoli.

Morbid Anatomy.—1. The *primary seat* of pulmonary phthisis is in the apices of the lungs.

2. The *histological changes* are—

(1) *accumulation of epithelial cells in the alveoli of the lungs* from desquamation of the alveolar epithelium ; the cells are granular and fatty from the action of the leucomaines ;

(2) the *exudation of fibrinous material and leucocytes into the alveoli* (see Inflammation) ;

(3) *cellular infiltration and thickening of the alveolar walls* and the walls of *terminal bronchioles* from the exudate of leucocytes and their conversion in part into connective tissue;

(4) *increase of inter-lobular connective tissue*, from the exudate of leucocytes and the formation of connective tissue therefrom.

3. The *extent* of these changes varies; usually at first only one apex is involved, but the disease spreads till a large part or the whole of one lung is involved, and later the other is affected. Usually a large *caseous mass* is formed, or in very chronic cases of slight intensity there is a gradual increase of connective tissue till the whole lung becomes greatly indurated (fibroid phthisis); these morbid conditions may be combined in different degrees.

4. The *bronchi* are more or less inflamed in the *catarrhal* form of phthisis, and in the *fibroid* form may be greatly dilated from the contraction of the new formed connective tissue.

Later Changes.—

1. The *caseous mass* may undergo one of three changes—

(1) *absorption*;

(2) *softening* and discharge, with the formation of a cavity;

(3) *calcification*—the calcified mass being surrounded by a wall of connective tissue.

2. In *fibroid phthisis* the gradual contraction of connective tissue causes *depression of the chest walls*, dilatation of the bronchi (*bronchiectasis*) and *displacement of neighboring viscera*. It results in *hypertrophy* and *dilatation of the right side of the heart* from the destruction of blood vessels in the lungs.

3. *Cavities* are formed by the softening and expectoration of caseous masses; *their walls* may be cheesy and rough or smooth and more or less firm; they are usually crossed by bands of connective tissue which contain blood vessels, and one or more bronchi project into them—being more resistant than the caseous mass; *the contents* of a cavity may be cheesy matter, pus or blood; the *tissue* around a cavity is consolidated and the *pleura* over it usually inflamed and thickened. *Cicatrisation* occasionally occurs after the contents of a cavity have been discharged.

Symptoms.—1. *Respiratory.* *Cough*, usually dry and hacking in the early stages and “loose” later on, occurs in all cases; it is probably due to the irritation of the bronchial mucous membrane.

Expectoration is scant at first and usually profuse at a later period in the caseous form. Scant in the fibroid form as a rule unless there is bronchiectasis. It is muco-purulent in character, often contains elastic fibers and nearly always contains bacilli.

Dyspnea is not usually marked except on exertion, because of the gradual loss of breathing space and the simultaneous loss of flesh.

Hæmoptysis is frequent; the blood comes from the bronchial

mucous membrane; it is scant and frothy in the early stages but later it may come from the rupture of a vessel in the cavity and be very profuse; it is bright red in color. *Hoarseness* and *loss of voice* may be present from laryngeal phthisis.

2. *Temperature*. More or less elevation of temperature is usually present in the early stages, the afternoon temperature being about 100° to 101° . At a later period there may be high fever from the absorption of septic matter contained in cavities. In such cases the fever is usually preceded by a chill and followed by sweating.

3. *Circulatory*. The pulse is quick and weak from the loss of strength.

4. *Nervous*. There is usually remarkable cheerfulness. Pain is not a prominent symptom, but may be present from accompanying pleurisy.

5. *Digestive*. *Anorexia* is marked, even in the early stages, and nausea and vomiting are frequent at a later period. *Diarrhœa* may occur from a complicating intestinal tuberculosis.

6. *Urinary*. *Albuminuria* may occur from tubercular nephritis.

7. *Cutaneous*. *Pallor* occurs early, as a rule, and is prominent. It is probably due to the action of the tubercle bacilli in preventing the formation of red corpuscles or actually destroying them. *Edema* occurs in the late stages from defective nutrition of the walls of the vessels and poor quality of blood, and sometimes from thrombosis of a vessel.

Night sweats are exceedingly common; no satisfactory explanation can be given of their occurrence.

8. *General*. *Emaciation* and *loss of strength* are usually progressive, and are due probably in part to the direct action of the germs and in part to the fever.

Physical Signs.—The *physical signs* depend on the form. In the *catarrhal* form there are *three* stages—1st, that of commencing formation of tubercles; 2d, that of consolidation (cascation); and 3d, that of cavity formation.

The *fibroid* form is far more chronic in course and does not usually lead to the formation of cavities. The physical signs of the two forms will therefore have to be considered separately.

A. Catarrhal phthisis.

1st stage—*Incomplete consolidation*.

1. On *inspection*, there may be (1) *slight depression* at the apex of the lungs from consolidation of some of the air cells at that point; (2) *less movement* than natural in respiration for the same reason.

2. On *palpation*, there may be *slight increase* in *vocal fremitus* at the apex because the lung is more solid than normal.

3. On *percussion*, *slight dullness* for the same reason.

4. On *auscultation*, (1) *broncho-vesicular respiration*; (2) *increased vocal resonance* from increase of solid matter in the lung; (3) *pilo-*

longed expiration, from loss of elasticity of air cells; (4) *crackling*, from slight pleuritic friction or from slight bronchitis.

2d stage—*Complete consolidation*.

1. On *inspection*, some changes as in 1st stage, but more marked.
 2. On *palpation*, *vocal fremitus increased*, unless (1) there is a layer of fluid between lung and the chest wall, (2) the bronchus leading to the consolidated spot is closed, or (3) there is a considerable thickness of healthy lung tissue over the consolidated spot.

3. On *percussion*, *dullness* of greater or less degree, because the lung is solid and contains little or no air.

4. On *auscultation*, (1) *bronchial breathing* unless prevented by the conditions which may prevent vocal fremitus (q. v.); (2) *râles* from bronchitis, pleurisy or breaking down of caseous mass.

3d Stage.—*Cavity*.

1. On *inspection*—same as in 2d stage.

2. On *palpation*, (1) *vocal fremitus increased* if layer of consolidated lung around the cavity, but may be absent; (2) *rhoncal fremitus* from the bursting of bubbles in the cavity.

3. On *percussion*, (1) *dullness*, if consolidated lung is around the cavity; (2) *cracked-pot* sound, if the cavity communicates with bronchus by a small opening; (3) *tympanitic resonance*, if the cavity has firm walls and the patient's mouth is open during percussion; (5) *flatness*, if the cavity is filled with fluid; (6) *normal resonance*, if healthy tissue surrounds the cavity.

4. On *auscultation*, (1) *Cavernous* or *amphoric breathing*, if the cavity is large and empty; (2) *cavernous whisper*; (3) *coarse bubbling* or *gurgling râles* from the passage of air through the fluid in a cavity.

B. *Fibroid phthisis*—

1. On *inspection*, (1) the *size* of the chest is more or less diminished; often it is greatly contracted; (2) the *movements* are much less free than normal; both changes are due to the formation and contraction of connective tissue.

2. On *palpation*, (1) the *vocal fremitus* is usually increased unless the pleura is greatly thickened or the bronchus is closed; (2) the *position* of the heart's beat may be changed from the change in size and position of the lungs.

3. On *percussion*, *dullness* over the lung when there is increase of connective tissue, and *increased resonance* or *tympanitic resonance* over other parts from compensatory emphysema.

3. On *auscultation*, *bronchial breathing* unless the pleura is greatly thickened or the bronchus is occluded.

Diagnosis.—The essential point in the diagnosis of tuberculosis from all other affections, is the presence in the sputa of the *baci li* of tuberculosis.

1. From *bronchitis* it is further distinguished by the fact that bronchitis occurs at the same time in both lungs, the râles are usually coarser and more extensively distributed, and there is no dullness on percussion.

2. From *pleurisy* it is distinguished by the *dullness (not flatness)* on percussion, and by the further fact that tuberculosis usually begins at the apex. (A *dry pleurisy*, however, is often tuberculous).

Prognosis.—The *prognosis* is always serious, but recovery may occur from (1) *calcification* of the tubercular mass, or (2) its *discharge* with subsequent healing of the cavity.

The *circumstances influencing the prognosis* are—

1. *Age.* Elderly persons usually have *fibroid* phthisis, which runs a much slower course than the catarrhal form.

2. *Heredity.* The more decided the hereditary taint the more serious the prognosis.

3. *Bad hygienic surroundings*, and *complications* render the prognosis grave.

Treatment.—A. *Prophylactic.*—1. *Children of a phthisical mother* should not be nursed by her, but by a healthy wet nurse, or they should be fed on *pure milk*.

2. *Young persons with phthisical tendencies* should live much in the open air, and should have a liberal diet.

B. *Hygienic.* Pure air, warm clothing and a nutritious diet are essential.

C. *Climatic.* The desirable features with respect to climate are (1) freedom from germs, (2) freedom from dampness, (3) sufficient warmth to allow the patient to spend much of the time in the open air.

The favorite resorts in this country are Colorado, the Adirondacks, California, Asheville, N. C., South Carolina, Florida, and Thomasville, Ga.

D. *Medicinal.*—1. To *improve the general health and sustain strength* by cod liver oil, arsenic, the hypophosphites and alcohol.

2. To *reduce fever* by phenacetine and quinine.

To *allay cough* by codeine, chloroform, and other agents of this class.

4. To *prevent night sweats* by atropia, ergot or agaricine.

5. To *relieve hæmoptysis* by ergot, cupping, absolute quiet, &c.

E. *Antiseptic treatment* has given poor results. Creosote, carbolic acid, iodoform, and bichloride of mercury have been given *internally*; of these, *creosote* seems to have done some good. *Inhalations* of creosote and carbolic acid have also been employed.

Injectons of iodine or carbolic acid into the lungs, and the *incision and drainage* of cavities have not given good results.

The use of *Koch's lymph* (tuberculine) has not up to this time

(April 2d, 1891) been followed by satisfactory results, so far as *treatment* is concerned, and the reaction from the treatment—chill and fever—is sometimes very severe, and has repeatedly caused death.

CHAPTER IV.

DISEASES OF THE DIGESTIVE SYSTEM.

STOMATITIS.

Definition.—By stomatitis is meant an inflammation of the mucous membrane of the mouth.

Varieties.—The varieties are—1. Catarrhal. 2. Ulcerative. 3. Aphthous or follicular. 4. Thrush.

CATARRHAL OR SIMPLE STOMATITIS.

Definition.—A superficial inflammation of the mucous membrane of the mouth characterized by redness and swelling.

Causes.—1. *Age.* The disease is rather more common in children than adults, but may occur at any age.

2. *Mechanical irritation* by broken teeth, &c., is a frequent cause.

3. *Chemical irritation* by alkalies or acids or by spices or hot substances will also cause it.

4. *Mercury* when improperly administered will induce the affection.

5. It occurs in *certain infectious diseases*, such as scarlet fever, small-pox, &c.

6. It may arise by *extension from neighboring parts*, such as the pharynx or nose.

7. *Defective* cleanliness of the mouth in insane persons, or those who are delirious, may cause it by the retention of pieces of food.

Symptoms and Signs.—1. *Sensory.* There is more or less pain in the mouth which is greatly increased by eating and especially by acids or sweets. Usually there is a very unpleasant taste in the mouth.

2. *Secretory.* The secretion of saliva is greatly increased and is often mixed with tenacious mucus. The increased secretion is due to the increased flow of blood to the mucous follicles and salivary glands.

3. On *inspection* the mucous membrane is found to be reddened from the increased amount of blood in it and swollen and softened from the serous exudate, so that frequently it shows the prints of the teeth.

Prognosis.—The *prognosis* of the affection is uniformly good and it rarely lasts more than a week or ten days.

Treatment.—The treatment consists in :

1. The *removal of the cause.*
2. *Thorough cleansing* of the mouth by frequent washing with boracic acid or some other similar agent.
3. The *relief of pain* by the use of small pieces of ice or the use of bland fluids such as milk or cream or some mucilaginous drink.
4. The *use of astringents*, such as myrrh in chronic cases.

ULCERATIVE STOMATITIS.

Definition.—An inflammation of the mouth characterized by the formation of ulcers usually on the gums and about the teeth.

Causes.—1. Probably *infection* is the essential cause. It is sometimes epidemic among soldiers and in prisons.

2. *Age.* It is most common, perhaps, among poorly nourished children.

3. *Debility* and bad hygienic surroundings predispose to it.

4. It occurs in severe *salivation* from mercury.

Symptoms and Signs.—1. *Seat.* The ulcers are usually situated on the gums.

2. *Pain* is often quite severe, especially when eating.

3. There is a copious *flow of saliva* and mucus.

4. The *glands* of the neck are usually considerably enlarged in consequence of the absorption by the lymphatics of the germs or leucomaines in the ulcers.

5. *Fever* sometimes occurs and *debility* is usually a marked symptom.

6. On *inspection* the gums look red and swollen; the teeth are often loosened, and ulcers with soft borders are seen.

Prognosis and Duration.—Such cases nearly always end in *recovery* in ten days or two weeks. Rarely the bones become diseased and the case is more tedious.

Treatment.—1. To insure *cleanliness* is of the first importance. Frequent washing of the mouth with diluted listerine (boracic acid) or salicylic acid or carbolic acid solution will accomplish this.

2. *Chlorate* of potash seems to be almost a specific in these cases; probably the chlorine it contains acts as a germicide; it should be used as a mouth wash and a *small quantity* may be swallowed.

APHTHOUS STOMATITIS.

Definition.—An inflammation of the mucous membrane of the mouth, characterized by the formation of white spots and subsequently ulcers, chiefly on the buccal surface.

Causes.—1. *Age.* It may occur at any age, but is more common in children than in adults.

2. It is *probably* due to a germ, but as yet none has been discovered.

Symptoms and Signs.—1. *Sensory.* The pain which these little spots cause is often excessive.

2. The *secretion* of saliva and mucus is usually increased.

3. On *inspection*, white spots varying in size from half a line to two lines in diameter are seen on the buccal mucous membrane or under the tongue. These spots consist of a fibrinous exudate in the most superficial layers of epithelium. After a time the white spots are replaced by a superficial ulcer.

Diagnosis.—The disease is distinguished from *thrush* by the absence of the parasite *mycoderma vini*, which is characteristic of the latter disease.

Prognosis and Duration.—The *prognosis* is always favorable and the disease rarely lasts longer than a few days.

Treatment.—1. To *relieve the pain* of these little spots, especially where an ulcer is formed, nothing is so efficacious as touching them with a stick of nitrate of silver.

2. *Cleanliness* and *disinfection* of the mouth by means of frequent washing with a solution of borax or boracic acid, or carbolic acid, is essential to prompt relief and cure.

THRUSH.

Definition.—An inflammation of the mouth, attended by the formation of white patches on the mucous membrane which are due to the presence of a parasite, the *mycoderma vini*. The disease is extremely frequent.

Synonyms.—Muguet, sprue.

Causes.—1. The *essential cause* is the parasite or fungus mentioned, the *mycoderma vini*.

2. *Age.* The disease is far more common among children than adults; indeed, it is *very rare*, except in infancy.

3. *Artificial feeding* and *defective cleanliness* are prominent causes.

4. It is far more common among *weakly* children than among vigorous ones.

Symptoms and Signs.—1. On *inspection*, white spots may be observed upon the buccal mucous membrane or that of the tongue or gums. The fungus develops first in the middle layers of the mucous membrane and may cause considerable ulceration.

2. *Pain* on eating or attempting to nurse is so great that the little patients are nourished with difficulty.

3. There is usually a considerable *flow of saliva* and mucus.

4. *Debility* is nearly always a prominent symptom in severe cases.

Prognosis and Duration.—Except in the case of very feeble children, recovery usually occurs in a week or two. Death occurs sometimes in the case of very weakly children.

Treatment.—1. *Thorough cleansing* of the mouth and the bottle (in the case of “bottle-fed” children), is of the first importance.

2. *Germicides*, such as boracic acid or carbolic acid in solution, are useful. Borax or boracic acid is most commonly used.

3. *Nourishing food* is essential in view of the debility which is nearly always present in these cases. Rectal alimentation may be resorted to in extreme cases.

NOMA, OR CANCRUM ORIS.

Definition.—A gangrenous affection of the jaws. It is very rare.

Causes.—The *essential cause* is probably a germ. It occurs in children usually and especially in those who are surrounded by bad hygienic conditions and are debilitated.

Symptoms and Signs.—The disease first appears as a little black spot on the inner side of one cheek. It rapidly spreads until nearly the whole cheek becomes gangrenous. The neighboring glands are greatly enlarged and there is high fever from the absorption of septic matters. Prostration is usually extreme.

Prognosis and Duration.—The disease nearly always terminates in death and rarely lasts longer than two weeks.

Treatment.—The treatment consists in destroying the diseased tissue by caustics; the use of antiseptics and the administration of nutritive food stimulants and tonics.

GLOSSITIS.

Definition and Frequency.—By glossitis is meant inflammation of the tongue; the disease is very rare.

Causes.—It may be caused by the sting of a bee or wasp, and occasionally it occurs without any obvious cause.

Symptoms. and Signs.—The chief sign is a *great swelling* of the tongue; it may be so great as to interfere with respiration; it usually projects from the mouth.

The swelling and tension cause great pain and swallowing is very difficult. There is a copious flow of saliva and the glands of the neck are enlarged.

Prognosis and Duration.—The *prognosis* is favorable and the disease rarely lasts longer than a few days.

Treatment.—When the tension is great free *incisions* should be made to relieve pain, and lessen the swelling. Painting with *cocaine* solution gives great relief.

The strength should be sustained by suitable diet.

PHARYNGITIS.

Definition.—An inflammatory affection of the mucous membrane of the pharynx and tonsils.

It may be acute or chronic.

Varieties of the acute form.—1. *Catarrhal* or *simple*.

2. *Follicular* tonsilitis.

3. *Abscess* of the tonsils.

CATARRHAL PHARYNGITIS.

Definition and Frequency.—An acute inflammation of the mucous membrane of the pharynx, the exudate being catarrhal in character. It is the common "sore throat."

Causes.—1. *Sudden chilling* of the body when overheated or exposure to a draft is the most common cause.

2. *Mechanical irritation*, such as operations upon the pharynx or injuries to it.

3. *Chemical irritants*, such as swallowing concentrated acids or alkalies.

4. *Acute infectious diseases*, such as scarlet fever and small-pox.

5. *Infective*. Severe attacks of "sore throat" sometimes occur among students who are dissecting or exposed to septic influences. (Hospital sore throat.)

Symptoms and Signs.—1. *Sensorv.* Pain is always present to a greater or less extent; it is often severe.

2. *Deglutition* is not only painful, but more or less difficult.

3. The *voice* is somewhat affected, having a muffled sound, "as if the mouth was full."

4. *Secretory*. The flow of mucus and saliva is at first diminished, but subsequently becomes decidedly greater than normal.

5. *Constitutional*. The *temperature* is usually elevated, in severe cases rising to 103° or 104°. Other constitutional symptoms are *headache*, *backache* and *aching* in the limbs.

On *inspection* the pharynx looks red and somewhat swollen, and is usually partially covered with a muco-purulent exudate.

Prognosis and Duration.—The *prognosis* is uniformly favorable and the attack usually ends in three or four days.

Treatment.—The treatment consists in—1. The *reduction of inflammation* by the administration of Dover's powder, hot foot baths, and hot applications to the throat, all of which dilate the vessels of the skin and lessen the congestion of the throat.

2. The administration of *chlorate of potash*, which probably acts as a germicide and destroys the germs which caused or keep up the inflammation.

3. The *relief of pain* by cocaine spray if the pain is intense, or by poultices to the neck in less severe cases.

FOLLICULAR TONSILLITIS.

Definition.—An inflammatory affection of the pharynx, in which the tonsils are chiefly involved, the inflammation being confined chiefly to the follicles of those glands.

Causes.—The causes are the same as those of simple catarrhal pharyngitis.

Symptoms.—The *symptoms* are also like those of acute pharyngitis, except that on the tonsils are seen little *white spots* which are due to the accumulation of secretion in the follicles. Sometimes the secretion is spread over the tonsil so as to look like a false membrane.

Diagnosis.—This white matter on the tonsils differs from a diphtheritic membrane, however, in being *readily removed*, and not fibrous in character.

The Prognosis and Duration are like those of pharyngeal catarrh.

The Treatment does not differ from that of acute pharyngitis.

ABSCESS OF THE TONSILS.

Definition.—*Frequency and synonym.* This affection, sometimes called *parenchymatous tonsillitis* or quinsy, is an inflammation of the tonsil glands leading to suppuration in their substance. It is of common occurrence.

Causes.—1. *Catarrhal pharyngitis.*

2. *Rheumatism* is supposed by many to be a cause.

3. Some people have *repeated attacks.*

Symptoms.—1. *Sensory.* The *pain* is extreme and *swallowing* is very difficult.

2. *Speech* is very much impaired on account of the difficulty of using the muscles of the palate.

3. There is a *secretion* of glairy mucus in large quantities.

4. *Constitutional.* The temperature is considerably elevated (103° or 104°), and there is great prostration. Headache and loss of appetite are also prominent symptoms.

5. *Inspection* of the tonsils is difficult on account of the inability of the patient to open his mouth well. The tonsils, usually one, but both may be involved, are greatly swollen; hard at first but soft when pus has formed. There is decided swelling on the outside also.

Prognosis.—The *prognosis* is usually favorable; suffocation has occasionally occurred from bursting of the abscess into the throat and the escape of pus into the windpipe.

Duration.—The abscess usually bursts or is ready to open in four or five days.

Treatment.—1. To *relieve pain, cocaine* in the form of spray is by far the most efficacious remedy. Poultices to the throat sometimes give partial relief. A spray of menthol, 20 parts, to olive oil, 80 parts, is highly spoken of.

2. *Cutting* into the abscess with a guarded bistoury is indicated so soon as softening occurs.

ENLARGED TONSILS.

Causes.—1. *Age.* Hypertrophy of the tonsils is far more common in children than in adults.

Symptoms and Signs.—The tonsils project, from their increased size, into the cavity of the pharynx.

Persons with enlarged tonsil usually *snore* loudly and the *voice* is rather husky. *Hearing* may be impaired from pressure on the posterior nares.

Prognosis.—The *prognosis* is always favorable as to life.

Treatment.—*Medical.* Iodine and ergot applied locally sometimes effect a cure.

Excision of the tonsils is the best treatment.

CHRONIC PHARYNGITIS.

Definition.—Chronic inflammation of the mucous membrane of the pharynx. It is an exceedingly common affection and is frequently called “clergyman’s sore throat.”

Varieties.—1. *Simple*, in which the mucous membrane looks somewhat swollen and thickened and blood vessels may be seen running over it.

2. *Granular*, in which the mucous membrane is not only thickened, but numbers of little granules, *enlarged follicles*, are to be seen upon it.

3. *Dry pharyngitis*, in which the membrane has a peculiar *glazed* look.

4. The *hypertrophic* form in which the pharyngeal glands are enlarged so as to form little protuberances on the posterior wall of the pharynx.

Causes.—1. *Climate.* A climate characterized by sudden changes of temperature is very apt to cause or keep up chronic pharyngitis. *Dampness* is especially injurious.

2. *Occupation.* Any occupation which exposes a person to *dust* of any kind is a cause. A sedentary life, especially in over-

heated rooms, is also a cause. Public speaking is such a common cause that one of its synonyms, clergyman's sore throat, is derived in this way.

3. *Alcohol* used habitually to excess is a very potent cause.

4. *Smoking* or staying in close rooms, such as inns, when others are smoking is a very common factor in the production of this disease.

Symptoms and Signs.—1. *Sensory.* Pain is not usually present, but a feeling of discomfort and tickling is almost constant in most cases.

2. The *voice* may be somewhat impaired, and sometimes decided hoarseness is present.

3. The *secretory* symptoms vary; the amount of secretion is usually slight and quite tenacious; occasionally dry, brown crusts may be seen on the posterior wall of the pharynx.

4. The *physical signs* on inspection were mentioned under varieties.

Diagnosis.—The *diagnosis* presents no difficulties.

Complications.—*Hearing* is occasionally affected from an extension of the inflammation to the eustachian tube, or pressure on the mouth of the tube by an enlarged gland. *Laryngeal* complications sometimes arise.

Prognosis.—The *prognosis* as to life is good; as to recovery not good, as a rule.

Treatment.—The *treatment* is directed to:

1. The *removal of the cause.* A change of climate when practicable is very useful. Alcohol and tobacco should be abandoned. Rest to the voice is essential.

2. The *removal of secretion* is effected chiefly by sprays; a solution of borax is very efficacious.

3. The *correction* of the pathological changes. When there are enlarged vessels on the posterior pharyngeal wall the local use of *iodine* and *ergot* is useful; a spray of *boracic acid* or of *carbolic acid* also acts well. Other agents are solution of *nitrate* of silver or *chloride* of zinc in water or *tannic acid* in glycerine.

In the *dry* form when stimulation is indicated a spray of *menthol* 20 parts to olive oil 80 parts is very useful. If there are *enlarged glands* they should be *excised* off or destroyed with Paquelin's or the thermo-cautery.

RETRO-PHARYNGEAL ABSCESS.

Retro-pharyngeal abscesses are rare, but are occasionally seen in children. The pus is situated between the posterior wall of the pharynx and the vertebral column.

The *symptoms* are difficulty in swallowing and after a time in breathing. On examination a soft swelling will be found at the back of the pharynx. The *prognosis* is serious unless proper treatment be adopted. The *treatment* is free incision.

DISEASES OF THE ŒSOPHAGUS.

Inflammation of the œsophagus is rare and of no clinical significance.

Obstructions of the œsophagus may be caused by—1. Spasm. 2. Stricture. 3. Cancer. 4. Foreign bodies. 5. Outside pressure.

Spasm usually occurs in hysterical women.

Stricture is rare; it is due, as a rule, to swallowing concentrated alkalies or acids. It occurs chiefly in early life, and is treated by methodical dilatation with œsophageal bougies.

Cancer usually occurs in advanced life and is marked by the usual cachexia. It is incurable.

Foreign bodies, such as false teeth, occasionally lodge in the œsophagus and require to be removed by surgical means.

Outside pressure may be due to solid tumors in the mediastinum or to aneurisms.

Pouches or *dilatations* of the œsophagus are rare. The pouches occasionally attain considerable size, and the food may accumulate there instead of passing into the stomach. It is subsequently regurgitated.

DISEASES OF THE STOMACH.

ACUTE GASTRITIS.

Definition.—An acute inflammation of the mucous membrane of the stomach.

Synonyms.—Acute gastric catarrh. Acute indigestion.

Causes.—1. *Debility, anæmia* and *fevers* which lessen the amount of gastric juice formed and weaken muscular movements, thus interfering with digestion and causing fermentation.

Chemical irritants, such as decomposing meats; unripe fruits; milk containing tyrotoxin; alcohol, spices, and certain poisons, as arsenic.

Morbid Anatomy.—1. *Contents of the Stomach*; sometimes food always tenacious mucus mixed with serous exudate, epithelial cells and pus cells.

2. *Appearance and structural changes.* *Redness*, from increased amount of blood; *swelling* from exudate of serum and cells. Albuminoid degeneration of epithelial and gland cells from inflamma-

tion. Sometimes extravasations of blood from bursting of blood vessels or hemorrhagic exudate.

Symptoms.—1. *Digestive.*—1. Anorexia. 2. Thirst. 3. Pain or discomfort and tenderness. 4. Nausea and vomiting of tenacious mucus. All these symptoms are probably due directly to the inflammation and the irritability of the nerves therefrom. 5. Constipation because the duodenum and common bile duct are usually involved and less bile is discharged into the bowel and there is consequently diminished peristalsis.

2. *Nervous.* The nervous symptoms are headache and mental depression; these are probably due to the formation of certain leucomaines in the stomach which are absorbed and exert an injurious influence on the nervous system.

3. The *temperature* is rarely elevated to much extent, but occasionally there is slight fever.

Diagnosis.—When fever is present it resembles mild cases of *typhoid fever* from which it may usually be distinguished by the absence of splenic enlargement and the greater intensity of the gastric symptoms in acute gastritis. Caution is always necessary, however.

Prognosis.—Except in cases of *poisoning* or in *young children* the prognosis is nearly always favorable.

Treatment.—1. To *remove irritating matter* from the stomach is the first indication if vomiting has not already occurred. Ipecac and apomorphia are the best emetics when any are necessary. *Calomel* to act upon the bowels is also advantageous.

2. *Rest to the stomach* as far as possible is essential; milk and lime water in small quantities are useful. Rectal feeding may sometimes be resorted to with advantage.

3. To *allay irritability* bismuth and lime water, tincture of iodine, hydrocyanic acid, morphia and other drugs are useful. *Mustard plasters* over the stomach are very efficacious.

CHRONIC GASTRITIS.

Definition and Synonyms.—A chronic inflammation of the mucous membrane of the stomach. It is called also *chronic gastric catarrh* and *inflammatory dyspepsia*.

Causes.—1. *Anæmia* and *debility* are predisposing causes of great importance. An insufficient supply of blood to the stomach causes a diminution in the amount of hydro-chloric acid secreted and weakness of the muscular coat. Debility, however produced, also causes weakness of the muscular coat. The diminution of hydro-chloric acid and defective movements of the stomach cause the food to ferment and to set up inflammation of the stomach.

2. In *gout and Bright's disease* there is a retention of excrementitious matters in the blood which may cause gastritis.

3. *Mechanical hyperæmia*, from disease of the liver, heart or lungs, causes gastritis, by interfering with the formation of hydrochloric acid and by interfering also with the proper nutrition of the mucous membrane of the stomach.

4. *Alcohol, spices* and highly seasoned food act as direct irritants.

Morbid Anatomy.—1. The *exudate* and *mucus* form a tenacious muco-purulent coating on the stomach.

2. The *structural* changes are most marked near the pylorus. *Pigmentation* is always present to a greater or less extent from the destruction of the red blood corpuscles thrown out and broken up at the spot. The mucous membrane always contains *more connective tissue* formed from white blood cells (see Passive Hyperæmia). The cells lining the tubules are usually in a state of fatty or granular degeneration, and the mucous follicles may be atrophied from pressure of the new connective tissue, or cysts may be formed from closure of their mouths. Sometimes the connective tissue grows very luxuriantly in spots, forming a "mammulated" appearance; occasionally superficial ulcers are formed.

Symptoms.—1. *Digestive.* *Nausea* and *vomiting* are frequent, especially in drunkards. The vomited matters are *acid* from the presence of lactic and butyric acids formed by fermentation. They contain much tenacious *mucus* and "*sarcine ventriculi*," and eructations are frequent. The fermentation which causes the formation of acid is due to (1) want of hydro-chloric acid, (2) the coating of the food with tenacious mucus which interferes with digestion.

There is usually little or no appetite and much thirst. The bowels are usually constipated.

2. *Cardiac.* Palpitation of the heart and a feeling of oppression are common, from distension of the stomach with gas and reflex action through the vagus.

3. *Nervous.* Headache, dullness and mental depression are conspicuous symptoms, and are probably due to the absorption of the products of fermentation or decomposition of the food in the stomach. *Pain* in the stomach is not usually very pronounced, but there is some tenderness on pressure.

Urinary. The urine is usually scant, alkaline, and contains phosphates, probably in consequence of the diminution in the quantity of normal acid generated by the stomach.

Diagnosis.—From *atonic dyspepsia* chronic gastritis is distinguished by the tenderness over the stomach and the presence of the causes of the latter affection. It should be remembered, however, that atonic dyspepsia, by causing fermentation of the contents of the stomach, will set up gastritis.

Prognosis.—The *prognosis* depends on the cause. If that can be removed recovery may be expected, and not otherwise.

Treatment.—The indications of treatment and means of fulfilling them are—1. *To remove the cause.* *Diet* is of the utmost importance. Alcohol and all highly seasoned food should be scrupulously avoided, and milk, eggs and bland soups should be used. Starchy food is injurious as well as sweet things, because they readily ferment.

Digitalis is useful in heart failure, and *saline cathartics* will temporarily relieve the mechanical hyperæmia to some extent.

2. *To allay irritability*, by means of sub-nitrate of bismuth, small doses of opium, hydro-cyanic acid and other sedatives. In very severe cases, *washing out the stomach* gives more relief than anything else.

3. *To improve the general health* by the administration of strychnine, iron, the vegetable bitters, &c. Life in the open air is of great service.

4. *To improve digestion* and relieve annoying symptoms, *hydrochloric acid* is useful in all cases. It prevents fermentation and promotes digestion directly. Strichnine is useful also in improving digestion by increasing the flow of gastric juice and stimulating the muscular coat.

To prevent the formation of gas, tincture of iodine, carbolic acid or salicylic acid may be employed.

For heartburn, magnesia and aromatic spirits of ammonia are useful. Alkaline mineral waters have a very beneficial effect in many cases.

CANCER OF THE STOMACH.

Frequency and Varieties.—The stomach is a frequent seat of cancer. The most common variety of the disease in this organ is scirrhus, but soft cancer also occurs.

Causes.—1. *Age.* The disease is very rare before forty and occurs most frequently after fifty.

2. *Sex* seems to have no influence in cancer of the stomach (Strumpell).

3. *Heredity* is an undoubted cause in a large proportion of cases.

Morbid Anatomy.—1. *Seat.* The pylorus and lesser curvature are usually involved.

2. *Characteristics.* The size of the tumor varies; it is usually hard, contracted and nodular, and in the later stages ulcerates.

Symptoms.—1. *Digestive.* *Vomiting* is common, the vomited matters frequently containing some blood, but copious hæmatemesis is rare. The amount of free hydrochloric acid in the gastric juice is greatly diminished, or *none* may be found.

2. *Nervous.* Pain is present in nearly all cases; it often comes on after eating, but may occur at other times; there is usually some tenderness.

3. The *general symptoms* are *emaciation, exhaustion* and *cachexia*, which is of great importance in diagnosis.

4. Usually a *tumor* can be felt, and unless this can be done the diagnosis must be doubtful.

Diagnosis.—It is diagnosed from *ulcer* by the existence of a tumor and the marked cachexia. *Hæmatemesis* is usually much greater in ulcer than in cancer.

2. From *chronic gastric catarrh* it is distinguished by the absence of a history pointing to this disease, the presence of a tumor, the cachexia and the character of the vomited matters.

3. It is distinguished from an *aneurism* when pulsation is communicated to the tumor from the artery behind it by lifting the tumor or pushing it to one side when the pulsation ceases.

Prognosis.—The *prognosis* is uniformly bad. The *duration* is from six months to three years.

Results.—Results are—

1. *Stenosis* of the pylorus and consequent dilatation of the stomach.

2. *Extension* to surrounding parts.

3. *Ulceration* into the peritoneal cavity, or outwardly through the abdominal wall (rarely.)

Treatment.—The *treatment* is chiefly palliative and consists in the administration of opiates or other analgesics and of bland food.

Surgical measures—removal of the pylorus and attaching the duodenum to the stomach or attaching the small intestine to a healthy part of the stomach have been practised, but with unsatisfactory results.

ULCER OF THE STOMACH.

Definition and Synonyms.—An ulcer through the mucous coat of the stomach, which may extend through the muscular and serous coats. It is sometimes called simple ulcer or round ulcer.

Causes.—1. *Age and sex.* It is most common in women between the ages of fifteen and thirty-five.

2. *Anæmia* and *chlorosis* are causative conditions

3. The *essential cause* is a digestion of the stomach wall by the gastric juice. The presence of alkaline blood prevents this in health, but if from any cause, as (1) injury, (2) hemorrhage, (3) plugging of a small vessel, the circulation is stopped at any point, digestion of that part occurs and an ulcer is the result.

Morbid Anatomy.—1. *Characteristics of the ulcer.* There is us-

ually but one which is nearly always on the *lesser curvature* of the stomach, is *round* in shape, from *half an inch* to *three inches* in diameter, and is funnel shaped—the wide end of the funnel being at the mucous surface, the narrow end at the peritoneal.

3. *Condition of the surrounding tissues.* The surrounding tissues are infiltrated and hard from the formation of connective tissue.

4. *Condition of the peritoneum.* The overlying peritoneum is thickened and has undergone an adhesive inflammation by which the stomach has become adherent to some adjacent organ—the pancreas, liver, colon, diaphragm, &c.

4. *Mode of healing.* When healing occurs cicatricial tissue is formed and there is very great contraction.

Symptoms.—1. *Nervous.* Pain and *tenderness* are among the most frequent symptoms of gastric ulcer. The pain is usually worse immediately after eating, and is often very severe. It is located in the region of the stomach, but often radiates to the back. The *tenderness* is usually over a limited area (the ulcer.)

2. *Digestive.* There are the usual symptoms of chronic gastric catarrh, but in addition there is in nearly all cases a history of one or more attacks of *copious hæmatemesis*.

Diagnosis.—1. From *Cancer of the stomach*, q. v.

2. From *chronic gastric catarrh* it is distinguished by the greater pain and tenderness and the *hæmatemesis* in ulcer.

3. From *gastralgia* by the occurrence of hæmatemesis and the time at which the pain usually appears after taking food.

In many cases, especially when there has been no hemorrhage, the diagnosis of ulcer of the stomach cannot be made with certainty.

Prognosis.—Death occurs in about 50 per cent of the cases; the causes of death are (1) hemorrhage; (2) exhaustion; (3) peritonitis.

Results.—1. *Healing* may occur with contraction.

2. *Stricture* of the pylorus occurs, and consequent dilatation of the stomach, if the cicatrix is at the pylorus.

3. *Abscesses* in the walls of the stomach occasionally occur.

4. *Hemorrhage* is extremely common, and may cause death.

5. *Perforation* may occur into the peritoneal cavity, pleural cavity, or into the intestinal canal; or the liver may be involved and an abscess result.

Treatment.—1. *Rest to the stomach* is absolutely essential. Rectal alimentation should be resorted to for some weeks, if possible; then *small* quantities of *peptonized milk* or *meat* may be given by the mouth.

2. To *allay irritability and relieve pain*, *subnitrate of bismuth* in teaspoonful doses three times a day, should be employed; *morphia* and *cocaine* are also useful; *phenacetine* and the allied substances

may also be used with advantage. In some cases, when feeding by the mouth is essential, *washing out the stomach* once a day is very beneficial.

3. To *check hemorrhage*, *opium* is the best remedy. Perfect quiet in the recumbent position should be enjoined.

DILATATION OF THE STOMACH.

Frequency.—Dilation of the stomach sufficiently great to cause trouble is rare.

Causes.—There are three classes of causes—1. *Obstruction* of the *pyloric* orifice, from—

- (1) simple stricture;
- (2) cancer;
- (3) outside pressure from tumors, &c.

2. *Increased pressure* within the stomach, from—

- (1) habitual over-eating;
- (2) fermentation and the formation of gas.

3. *Weakness of the muscular walls* of the stomach, from—

- (1) chronic gastric catarrh;
- (2) general debility.

Morbid Anatomy.—1. *Situation* of the *stomach*. Nearly always the stomach is dragged down lower than natural in the abdominal cavity by its own weight.

2. *Size*. The size of the organ is greatly increased, and it may hold as much as seven pints of fluid. (Fagge).

Symptoms and Signs.—1. *Digestive*. The digestive symptoms are, in the main, those of chronic gastric catarrh and the causative affection. The vomiting of very large quantities of liquid with pieces of food taken some days before is characteristic.

2. On *inspection*, *palpation* and *percussion*, the lower border of the stomach is found below the umbilicus, and there is a marked fullness on the left side of the abdomen.

Diagnosis.—The *diagnosis* is based on :

1. The situation of the stomach.
2. The *size* of the stomach as measured by (1) the amount of fluid which is vomited out or can be pumped into it; and
- (2) the distance to which a stomach tube will enter; the tube passes usually in the case of a *normal* stomach *two feet* from the lips.

Prognosis.—The *prognosis* depends upon the cause. In *cancer* it is of course unfavorable. A *simple* stricture may *sometimes* be dilated, *inside pressure* may be lessened or removed and the walls may be toned up. The prognosis is doubtful, however, in all cases.

Treatment.—1. To *remove* the cause when practicable is the first thing to do.

2. To *wash out the stomach* and thus remove fermenting substances is more useful than anything else for obvious reasons.

3. Strychnine, ergot and electricity have been used to *tone up the muscular coat* and cause contraction of the stomach.

GASTRALGIA.

Definition.—*Synonyms and frequency.* By *gastralgia* is meant pain in the region of the stomach, usually occurring in paroxysms of great severity and not connected with inflammation or other local condition.

Causes—It is most frequent in young *women*, and especially in those of nervous temperament. Sometimes it seems to be due to cold. Anæmia seems to be a cause.

Symptoms.—There is no *digestive* symptom. The *pain* usually occurs in paroxysms often weeks or months apart, but sometimes every day or several times a day. The pain is sharp and lancinating in character and may extend to the back. It has no connection with eating. Usually there is marked anæmia, and menstrual disturbances are common accompaniments.

Diagnosis—It is distinguished from (1) *ulcer*, by the absence of vomiting (as a rule) and especially by the hæmatemesis; from (2) *cancer*, by the absence of cachexia and the age of the patient; from (3) *gastric catarrh*, by the absence of vomiting or other digestive disturbances.

Prognosis.—The *prognosis* is good.

Treatment.—The *treatment* consists in improving the general health by cod liver oil, arsenic, strychnine and iron, and the administration of analgesics during the paroxysms. Hot applications sometimes give relief.

HÆMATEMESIS.

Definition.—The vomiting of blood which was poured out in the stomach.

Causes.—1. *Ulcer and cancer*, q. v.

2. *Obstruction to the portal circulation*, as in cirrhosis of the liver.

3. The *hemorrhagic diseases*, purpura, &c.

Symptoms.—If in large quantity and thrown up soon after it is poured out the blood is *bright* in color.

If retained some time it looks like *coffee grounds*.

Diagnosis.—It is distinguished from *nose bleeding* or *bronchial hæmorrhage* by an examination of those parts.

Prognosis.—The *prognosis* depends entirely on the cause.

Treatment.—The *treatment* also depends in great measure upon the cause, but quiet is essential in all cases. Opium, gallic and tannic acid, ergot, turpentine and persulphate of iron have been given to check the hæmorrhage.

DISEASES OF THE INTESTINES.

DIARRHŒA.

Definition and Frequency.—An abnormal frequency of the discharges from the bowels, which are nearly always less consistent than in a state of health. It is of very common occurrence.

Causes.—There are two *general* causes of diarrhœa.—1. An *excessive secretion* or transudation of liquid into the bowels.

2. *Excessive peristalsis* of the bowels, which forces out their contents before the liquid portion can be absorbed.

Excessive secretion may be due to—1. Nervous influence, as fright or anxiety.

2. *Inflammation*.

3. The action of certain drugs (saline cathartics).

Excessive peristalsis may be due to—1. *Nervous influence*, irrespective of inflammation.

2. *Inflammation*, and increased reflex irritability in consequence.

Symptoms.—The *symptoms* attendant on diarrhœa need but little notice here. The *character* of the discharges depends on the cause of the attack and the part of the bowel affected.

Other matters in connection with diarrhœa will be considered in connection with the different diseases of which it is a symptom.

ACUTE INTESTINAL CATARRH.

Definition, Synonyms and Frequency.—An acute inflammation of the mucous membrane of the intestines. It is of very common occurrence, and is known as "*enteritis*," "*catarrhal enteritis*," "*acute diarrhœa*," &c.

Causes.—1. *Season of the year.* Attacks of acute diarrhœa are more frequent in the summer and autumn than at other seasons, probably because most germs find favorable conditions for development then and because the diet at those seasons is liable to be irritative in character.

2. *Improper food and drink*, such as unripe fruit, diseased meat, impure drinking water, &c., are common causes; the irritating substances which they contain are apt to cause inflammation.

3. *Exposure to cold and dampness* or to *extreme heat* may cause acute intestinal catarrh. Cold and wet probably act by forcing the blood from the surface. The action of heat is not understood: it is probable that it only acts *indirectly* by facilitating the growth of germs and the formation of noxious substances in the food and water (Fagge).

4. *Constipation* often alternates with diarrhœa and causes the latter condition by irritating the mucous membrane of the bowels.

5. The *eruptive fevers* often cause diarrhœa. (See *Acute Infectious Diseases* for explanation).

Morbid Anatomy.—1. *Seat and extent* of bowel involved. Usually a catarrhal enteritis affects the *lower part* of the *small intestine* and the upper part of the *colon*. *Diarrhœa* occurs in such cases.

The *duodenum* alone or the duodenum and adjacent parts may be involved; in such cases diarrhœa does not occur and jaundice is a frequent complication.

2. *Structural changes*. The usual changes in inflammation of a mucous membrane are present—redness, swelling, enlargement of the glands, albuminoid degeneration of the lining cells, ulceration in severe cases.

3. *Secretion and exudate*. The secretion of mucus is increased from increased afflux of blood and from mucoid degeneration of the cells and this is mixed with the *serous* exudate and cells which have passed out of the vessels and the epithelial cells which have desquamated. Rarely in very severe cases the exudate may be *fibrinous* in character and lead to more or less extensive *necrosis* of the mucous membrane.

Symptoms.—1. *Digestive*. *Diarrhœa* is the most striking symptom in those cases which involve the colon. It is due to (1) increased secretion and exudate and (2) increased irritability of the nerves leading to more rapid peristalsis. The discharges are liquid in character and contain no mucus which is separate from the fecal matter.

Constipation occurs in those cases where the inflammation is limited for the most part to the duodenum because the flow of bile is impeded and there is consequent diminution of peristalsis. More or less jaundice is common in such cases.

Nausea may occur from absorption of morbid products or from a simultaneous affection of the stomach. *Flatulence* from decomposition of food is of common occurrence.

2. *Nervous*. *Pain and tenderness* are present in nearly all cases, but are rarely severe. The pain often comes on just before an action on the bowels and the patient is easy in the intervals.

3. *Temperature*. The fever is rarely high, but as a rule there is some elevation of temperature.

Diagnosis.—1. From *dysentery* enteritis is distinguished by (1) the absence of tenesmus; (2) the absence of free mucus or blood in the actions.

2. From *poisoning* it is differentiated by the history of the case and the greater gravity of the symptoms as a rule in cases of poisoning.

3. From *typhoid* fever by the absence of an enlarged spleen and the difference in the course of the fever.

Prognosis.—The prognosis is nearly always good in acute enteritis.

Complications.—1. *Jaundice*.

2. Inflammation of the stomach.

Treatment.—1. The *diet* should be mild and unstimulating. Rest in bed is important for speedy recovery.

2. To *remove offending matters* when present, purgatives, such as calomel or the salines, are useful.

3. To *relieve pain*, to *allay inflammation* and to *check discharge*, opium and bismuth, salol and the alkalies, such as bicarbonate of soda or aromatic spirits of ammonia are the best remedies. After the acute stage has passed, astringents, tannic acid, catechu, kino, alum, &c., may be employed to check discharge.

PHLEGMONOUS ENTERITIS.

Definition and Frequency.—A rare disease characterized by inflammation of serous and muscular coats of the intestines.

Causes.—*Intussusception* and strangulation of the bowels are the most usual causes. Rarely there is no obvious cause.

Morbid Anatomy.—Extreme congestion and discoloration of the serous coat, along with softening and serous exudation into the muscular coat, constitutes the morbid anatomy.

Symptoms.—Extreme pain and tenderness, nausea, vomiting, tympany and obstinate constipation, due to inflammation and consequent loss of tone of the muscular coat.

Diagnosis from peritonitis is impossible; indeed *it is a localized peritonitis*.

Prognosis.—Serious.

Treatment.—*Opiates*, to relieve pain; *saline purgatives*, to empty the bowel, unless there is obstruction, when laparotomy is necessary.

CHRONIC INTESTINAL CATARRH.

Definition, Synonyms and Frequency.—A chronic inflamma-

tion of the mucous membrane of the intestines. It is called also "chronic diarrhœa." It is of frequent occurrence.

Causes.—1. Frequent attacks of the acute form.

2. *Passive hyperemia*, as in cirrhosis of the liver and certain heart troubles.

3. *Tuberculosis* of the bowels.

4. *Waxy degeneration* of the bowels.

Morbid Anatomy.—1. In the *simple* form, (not tuberculous or lardaceous), there are the usual evidences of chronic inflammation of a mucous membrane, shaven beard appearance or pigmentation, increase of connective tissue with either atrophy of the mucous membrane or the formation of polypoid growths. Little cysts are sometimes formed from closure of the mouths of the follicles and the accumulation of secretion in them.

2. In the *tuberculous* cases the glands, solitary and agminated in the lower end of the ilium and upper end of the colon, are involved chiefly. Tubercles (q. v.) developed in them undergo caseation and softening, and an ulcer is formed with infiltrated edges and which extends around the bowel. The mesenteric glands in these cases are nearly always enlarged and often caseous; they may soften and burst or calcify.

In *waxy degeneration* the changes are not marked. The mucous membrane of the intestine may look rather translucent and œdematous, and if iodine be poured over it a mahogany color is produced.

Symptoms.—1. *Digestive.* *Diarrhœa* is the most common of the digestive symptoms.

2. *Nervous.* *Pain* is not usually present, or, at any rate, is not a conspicuous symptom.

3. In the *tuberculous* form *emaciation* is progressive and sometimes becomes extreme. There are usually symptoms of tuberculosis elsewhere also.

4. In cases of *lardaceous* degeneration in addition to the *diarrhœa* there are usually evidences of waxy degeneration of the liver and kidneys.

Diagnosis.—The *diagnosis* as to the cause of chronic diarrhœa is based on the associated affections, such as cardiac or hepatic trouble, tuberculosis of the lungs or waxy degeneration of the liver or kidneys.

Prognosis.—The *prognosis* depends upon the cause, but is usually very serious.

Treatment.—1. To *remove the cause* if possible is the first indication.

2. *Diet* and *dress* are very important; the diet should be bland

and nutritious (milk, eggs, &c.,) and flannel should be worn next the skin to avoid chilling.

3. To *check the discharge*—bismuth, tannic acid, persulphate of iron, nitrate of silver, opium and many other remedies have been employed and may be tried with hope of amelioration if not cure.

CHOLERA MORBUS AND CHOLERA INFANTUM.

Definition and Frequency.—A disease characterized by profuse vomiting and purging, great prostration, and in adults by cramps in the legs, &c. It is quite common.

Causes.—1. *Season.*—It is far more common in hot weather than in cold.

2. *Fatigue and debility* are predisposing causes.

3. *Sudden chilling* of the body when over-heated may induce an attack.

4. *Improper food, especially in the case of children*, is a very prominent cause.

Attacks of milk poisoning or ice cream poisoning are due to the formation of tyrotoxicon in the milk by the action of germs (Vaughan).

It is probable that the essential cause is always a germ.

Morbid Anatomy.—There is generally no morbid change apparent. Gastro-enteritis is occasionally found.

Symptoms.—1. *Digestive.* Sudden and violent vomiting and purging at first of the contents of the stomach and bowels and then of liquid. Great thirst.

2. *Nervous.* Pain in the belly and cramps in the legs are always present in adults.

3. *Circulatory.* The pulse becomes rapid and weak and may be imperceptible; the skin is cold and clammy.

3. *General.* Prostration is extreme, especially in cases of cholera infantum and the anterior fontanelle in the case of infants is sunken.

Diagnosis.—1. From *Asiatic cholera* it is distinguished by the absence of the comma bacillus from the discharges and the sporadic character of the disease.

2. In *irritant poisoning* the symptoms are usually more persistent than in cholera morbus, and blood is vomited, and discharged by the rectum.

Prognosis.—The *prognosis* in the case of adults is nearly always favorable.

In children the disease (called cholera-infantum in them) is extremely dangerous.

Treatment.—In *adults*, counter-irritants, hypodermic injections of morphia and stimulants should be employed.

In *children*, counter-irritants, stimulants, heat to the extremities, and lastly *fresh air*, are of great importance.

Cholera infantum is very liable, if the immediate danger is prevented, to run into a chronic catarrh of the intestinal canal.

INTESTINAL CATARRH OF CHILDREN.

Definition, Synonyms and Frequency.—An inflammatory affection of the mucous membrane of the bowels. It is also called enterocolitis and sometimes improperly cholera infantum. It is of great frequency, especially in crowded cities and in hot weather.

Causes.—1. The *essential cause* is probably a germ or a number of germs of different character.

2. *Age.* It occurs usually in children between the ages of six and eighteen months, but may occur earlier or later. The influence of age is explained by (1) the fact that before six months most children use breast milk and (2) after two years of age the tissues of the intestines seem to be more resistant than they are at an earlier period.

3. *Heat.* The vast majority of cases occur in hot weather for two reasons. A temperature of 60° throughout the day greatly facilitates the action of germs and the formation of leucomaines (tyrotoxicon) and furthermore heat is terribly depressing to the child and renders its tissues less resistant.

4. *Overcrowding* is a potent factor because it causes debility and renders the child an easier prey to any disease.

5. *Improper food* is the most direct and common cause. Milk which has undergone a change resulting in the formation of *tyrotoxicon* is in the majority of cases responsible for the disease.

Morbid Anatomy.—1. *Seat and extent.* The part of the bowel usually involved is the lower part of the ileum and the upper part of the colon. This is probably due to the fact that there is a partial stagnation of faecal matter in this part of the intestine so that the germs have an opportunity to do mischief there. (See lecture on Bacteria.)

2. *Contents of the bowel.* The contents of the bowel consist of faecal matter, mucus, epithelial cells, pus cells and serum, sometimes blood. The mucus in the large bowel is often very tenacious.

3. *Structural changes.* The vessels are distended and the mucous coat of the bowel is swollen in consequence of the exudate of

serum and leucocytes. The follicles are enlarged and softened and may be pigmented; at a late stage they ulcerate.

Symptoms.—1. *Digestive.* *Diarrhœa* is the first and most prominent symptom; the actions vary in number and character. They may be white from absence of bile; they may be green; they often contain little white masses of either fat or casein; rarely they contain blood and mucus (dysenteric stools.) *Tenderness* is almost always present to some extent and *pain* is a common symptom. *Tympanites* frequently occurs. *Vomiting* is usual and often persistent. *Thirst* is sometimes a very troublesome symptom. The *tongue* is coated with a white fur and is red at the tip and edges in the early stages, but if exhaustion comes on it becomes brown and dry.

2. *Nervous.* *Sleeplessness* and *restlessness*. At a later stage *convulsions* and *stupor*. Pain in the belly has already been mentioned.

3. *Circulatory.* The pulse is rapid and soon becomes feeble; the skin is pale and in protracted cases there is swelling of the feet and ankles from cardiac weakness.

4. The *temperature* is elevated from 101° to 104° ; but if prostration occurs it may fall below normal (collapse.)

5. *General.* *Pallor*, *emaciation* and *prostration* are due to the loss of nutritive material in consequence of the diarrhœa and the defective absorption by the inflamed mucous membrane.

Diagnosis.—The *cerebral* symptoms may be mistaken for those of acute hydrocephalus. In the latter disease the bowels are constipated, the face flushed usually and the anterior fontanelle prominent; the reverse is the case in infantile entero-colitis.

Prognosis.—The *prognosis* depends, in great measure, on the possibility of moving the child to the country and changing its food supply. The disease is always serious and prompt measures are necessary.

Treatment.—A. *Prophylactic.* Pure air, pure milk and proper hours of feeding are best prophylactics. The child should be taken to the country if possible. The *milk* (when bottle fed) should be *sterilized*.

B. *Remedial.* The objects of treatment are—1. To *destroy germs* and *check fermentation* by calomel, bismuth, salol, naphthalin, resorcine, &c.

2. To *remove morbid matters* from the stomach and bowels by purgatives (*rarely advisable*) and *irrigation* of the stomach (?) and *bowels*.

3. To *allay pain* and *restlessness* by opium and paraldehyde and by warm or cool baths.

4. To *check discharge* by bismuth, opium, catechu, &c.

5. To *sustain strength* by stimulants and proper food.

TYPHLITIS AND PERI-TYPHLITIS.

Definition, Synonym and Frequency.—An inflammation in and around the cæcum and vermiform appendix. It usually commences in the latter part and is frequently called "*appendicitis*." The disease is not very common.

Causes.—1. *Ulceration or bursting of the vermiform appendix* from—

- (1) the irritation of a foreign body ;
- (2) impacted and hardened feces ;
- (3) tubercle ;
- (4) distension from catarrhal inflammation ;
- (5) possibly injuries.

2. Rarely the disease begins as inflammation of the cæcum.

Morbid Anatomy.—1. *Changes in the appendix* are either ulceration or distension with catarrhal exudate (Fitz).

2. *Changes in the surrounding tissue* are—

- (1) localized peritonitis, with or without peritoneal abscess ;
- (2) inflammation with serous or purulent exudate in the surrounding connective tissue.

3. *Changes in the muscular and mucous coats* consist in infiltration with fluid and cells.

Symptoms.—1. *Digestive and nervous.* Pain and constipation are the prominent symptoms. The pain may not be felt at first in the region of the cæcum, but tenderness soon appears. Nausea and vomiting are frequent.

2. The *temperature* is elevated from 100° to 104° or 105° .

3. *General.* Chills, fever and sweats often occur, if pus is forming. Exhaustion and emaciation also occur under similar circumstances.

Physical Signs.—1. On *inspection*, the thigh on that side is frequently found to be *flexed* to prevent pressure and pain.

2. On *palpation* and *percussion*, dullness and a tumor may be detected in the right inguinal region.

Diagnosis.—The *diagnosis* is based on (1) the history of the case, (2) the seat of the swelling, (3) the existence of fever.

Prognosis.—About 74 per cent. recover (Fitz). The chief cause of death is (1) peritonitis from ulceration of the appendix *directly* into the peritoneal cavity, or the formation of an *abscess* which bursts into it, (2) exhaustion.

Results.—*Resolution* occurs in about 26 per cent. of cases treated medically.

2. The appendix may *ulcerate* directly into the peritoneal cavity.

3. *Abscess* may form, and if not opened, may burst (1) into the abdominal cavity, (2) into the intestinal canal, (3) into the bladder or pelvis of the kidney, (4) outwardly through the abdominal wall.
4. *Recurrence* occurs in about 40 per cent. of the cases.

Treatment.—1. *Medical.* Rest, opiates, poultices, enemata.
 2. *Surgical.* Laparotomy and removal of appendix in case of urgent symptoms with or without a tumor.

INTESTINAL PARASITES, OR WORMS.

Varieties.—The most important are—1. Tape, or *tænia solium*.
 2. Round, or *ascaris lumbricoides*. 3. Thread, or seat worms.

Tape and round worms are found in the small intestine; thread worms in the rectum.

Causes.—1. *Age.* Most common in children; tape worms occur in adults.

Modes of Infection.—1. *Meat or vegetables* (uncooked).

2. *Impure water*, containing ova of worms.

Symptoms.—1. *Anæmia and debility* (rare symptoms) from the withdrawal of blood by parasite (*anchylostoma duodenalis*).

2. *Obstruction of bowels* (rare).

3. *Local irritation*, pain, diarrhœa, &c., frequent symptoms.

4. *Reflex*—convulsions, strabismus, &c., not common.

Diagnosis.—Based on *sight* of the worms; round worm eight to twelve inches in length; thread worm three-quarter inch or one inch; tape worm, in flat segments, one-third to three-quarter inch in length.

Prognosis.—Usually favorable. Death may occur from (1) obstruction, (2) entrance into larynx, (3) convulsions.

Treatment.—A. *Prophylactic.*

1. Pure water.

2. Clean utensils and cleanliness generally.

3. Thorough cooking.

B. *Medicinal.*

For *tape* worms, kousso, pomegranate, male fern, chloroform, croton oil and pumpkin seed.

For *round* worms, santonin, calomel and turpentine.

For *thread* worms, enemata of quinine, aloes or gentian.

TRICHINOSIS.

Definition.—An affection characterized by digestive disturbances and great pain and stiffness of the muscles, the symptoms being due to the action of a parasite—the *trichina spiralis*.

Causes.—The *only cause* is the ingestion with raw or very rare meat, (pork) of the *trichina spiralis*.

Morbid Anatomy.—1. The *intestines* are usually inflamed by the action of the parasite which is liberated when the pork is digested.

2. The *parasite* is about one twenty-fifth of an inch long when first liberated, but grows to about one sixth of an inch in length and then discharges a great number of living young; the young parasites pass out of the bowel and reach the muscles.

3. The *muscles* most affected are the diaphragm, the muscles of the throat and the intercostal muscles. They are inflamed and many parasites are found in them coiled up and surrounded by an oval capsule formed chiefly of connective tissue.

Symptoms.—1. *Digestive disturbances.* Pain and diarrhœa are common.

2. *Nervous and muscular.* Violent pain and stiffness in the muscles and sometimes paralysis occurs.

3. The *temperature* is elevated to 101° or 106° .

4. There is *sweating* and *œdema*.

Diagnosis.—The *diagnosis* is based on an examination of a small piece of muscular tissue obtained by cutting or harpooning.

Prognosis.—The *prognosis* is uncertain.

Treatment.—*Prophylactic* is most important. Glycerine, picric acid and other remedies have been used, but with little success.

CONSTIPATION.

Causes.—1. *Defective power* of the muscular coat of the intestines from (1) debility; (2) want of nerve power (3) chronic catarrh of the bowels; (4) chronic peritonitis.

2. *Deficient secretion* from (1) want of proper nerve power; (2) atrophy of mucous follicles (simple chronic catarrh); (3) abuse of purgatives.

Morbid Anatomy.—1. *Dilatation* of the bowel from distension with fecal matter and gas.

2. *Ulceration* of the bowel from contact with hardened or decomposing fecal masses.

Accompanying Symptoms.—1. *Pain* in the bowels and *flatulence*, from irritation of the bowels by hardened feces and decomposition and fermentation of retained masses.

Furred tongue and *bad taste* in the mouth, from absorption of excrementitious substances.

2. *Nervous* disturbances—headache, dullness, stupor, &c., from retention of morbid matters.

3. *Respiratory* disturbances, from pressure of distended bowels on the diaphragm, and possibly from reflex action and influence of retained leucomaines.

4. *Cardiac* and *circulatory* disturbances, caused just as the respiratory.

Prognosis.—Good, unless the cause cannot be removed, and the case is of very long standing.

Treatment.—*Diet.* Branny food, fruits, vegetables and water, (especially before breakfast), to increase secretion.

2. Exercise, electricity, massage, &c., to increase the power of the muscular coat.

3. Certain medicines (*to be avoided if possible*), such as (1) strychnine, and possibly ergot, to strengthen the muscular coat, and (2) aloes, cascara, salines, belladonna, &c., to increase the secretion.

4. Enemata of water, &c., to move the bowels mechanically (not advisable).

Enemata of glycerine, or glycerine suppositories, to cause a flow of water into the lower bowel and to set up slight irritation (very useful).

DYSPEPSIA, OR INDIGESTION.

Definition and Frequency.—A disturbance of the function of digestion without obvious pathological change in the digestive organs.

Causes.—1. Anything which impairs the *quality* of the digestive fluids, such as (1) anæmia and debility, (2) defective nervous action, (3) sedentary habits.

2. Anything which impairs the *power of the muscular coat* of the bowels, such as those conditions just named, and in addition (1) the pressure of tumors, (2) adhesions and sclerosis of the bowels from former inflammation, (3) torpor of the liver, or *deficiency* of the bile, from any cause.

Symptoms.—1. *Digestive*, such as (1) heart-burn, (2) distension with gas, (3) regurgitation of food from fermentation and the formation of acids and gases, (4) sometimes vomiting from irritation of the stomach by fermenting food, (5) constipation from weakness of muscular coat of the bowels, or (6) diarrhœa from the irritation of fermenting food catarrh.

2. *Nervous* disturbances, such as (1) sleepiness, (2) stupor, (3) headache, (4) disturbances of vision, &c., from the absorption of leucomaines, and probably also from defective action of the liver, which usually acts as a filter.

3. *Cardiac and respiratory* disturbances, from pressure on diaphragm by distended bowels.

4. *Urinary*. Deposit of phosphates and oxalates from defective nutrition and want of acid.

Diagnosis.—1. Distinguished from *Gastric catarrh*, by the absence of stringy mucus in the vomited matter and the absence of tenderness.

2. *Cardiac disease*, by temporary character of symptoms and absence of physical signs.

Results.—*Chronic gastric catarrh*, unless dyspepsia is relieved.

Prognosis depends on cause and duration—usually good.

Treatment.—1. To stimulate secretory and muscular coats of the stomach and bowels by (1) exercise, (2) massage, (3) strychnine, (4) bitters, (5) alcohol, (6) emetics, &c.

2. To supply substances for formation of *gastric* juice, such as *pepsin* and *hydrochloric acid*, or of pancreatic secretion, such as extract of pancreas and bicarbonate of soda.

3. To relieve special symptoms, such as—

(1) heart-burn, by ammonia and alkalies;

(2) flatulency, by peppermint, or camphor and ammonia, for immediate relief, or by tincture of iodine, salicylic acid, carbolic acid, salol, &c., when a more lasting effect is desired.

(3) constipation, q. v.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum.

Varieties.—1. Local. 2. General.

Causes.—1. Diseases of some *abdominal* viscus, such as (1) ulcer of the stomach, or duodenum, (2) ulceration of the bowels, as in typhoid fever, (3) typhlitis and appendicitis, leading to perforative ulceration. If perforation of the gut does *not* occur the inflammation will probably be *adhesive* and localized; if perforation *does* occur it is usually purulent and generalized, because fecal matters and germs pass into the peritoneal cavity in two large amount to be disposed of by leucocytes; (4) intestinal obstruction.

2. Disease of some *pelvic* organs, such as salpingitis, &c., in which germs may pass along the genital canal and tubes to the peritoneal cavity, or a pyo-salpinx may burst into the peritoneal cavity.

3. *Extension of localized peritonitis rarely* causes an attack of generalized form.

4. *Injuries*, which admit germs to the peritoneal cavity.

5. *Certain acute, infectious diseases*, such as erysipelas, septicæmia, diphtheria, &c., and—

6. *Certain chronic diseases*, as *Bright's*: in both the latter classes of affections the disease is probably due to the circulation of morbid materials in the blood and lymph vessels. *Rheumatism* is an occasional cause.

Morbid Anatomy.—1. *Redness* and *loss of lustre* occur from the increased amount of blood in the vessels, and albuminoid degeneration of the endothelial cells. Desquamation of endothelial cells occurs from loss of their vitality and the action of the serous exudate.

2. The *exudate* may be *serous*, purulent, fibrinous or hemorrhagic; in severe cases it is chiefly purulent; in the localized form fibrinous and adhesive.

3. The *muscular coat* is swollen from the exudate of serum and leucocytes, and there is consequent loss of tone and tympanitic distention.

4. If recovery occurs there is great *increase* of *connective* tissue and more or less extensive *adhesions*.

Symptoms.—1. *Onset* usually sudden because of ulceration of a hollow viscus and sudden discharge of its contents; it may be gradual.

2. *Position* in bed—on the back, with limbs flexed on the abdomen because of—

3. *Pain* and *tenderness*, which is due to inflammation. No mental disturbance usually.

4. The *digestive* symptoms are (1) vomiting, probably reflex, (2) tympanites, and (3) constipation, from loss of tone of the muscular coat of the bowels.

5. The *temperature* is elevated (from 101° to 105°), especially in septic cases from absorption of leuconaines.

6. *Respirations* shallow and rapid, from distension of abdomen and pain caused by rubbing of peritoneal surfaces against each other in deep inspiration.

7. The *pulse* is small and hard and rapid, from reflex action through the vagus probably, but the cause is not clear.

8. *Frequent desire* to *pass* water from reflex irritation, and later *retention* from inflammation and loss of power of the muscular coat of the bladder.

Diagnosis.—It is distinguished from (1) colic, by fever and the increase of tenderness on pressure in peritonitis; from (2) enteritis, by constipation and excessive tenderness.

Prognosis.—Very bad in the generalized form; usually good in localized and adhesive form, which is protective.

Results.—1. *Death* in a large proportion of cases from (1) shock, (2) exhaustion, (3) septic poisoning.

2. *Recovery* occasionally in purulent form after (1) absorption of pus, (2) encapsulation and calcification, (3) discharge externally through umbilicus, vagina, bowel, bladder, &c.

Treatment.—1. To give *rest to the bowels* and relieve pain by (1) avoidance of purgatives, (2) opium, (3) belladonna, (4) turpentine stupes, (5) poultices.

2. To *sustain strength*, by rectal enemata when the stomach is intolerant.

3. *Laparotomy* and washing out abdomen with closure of opening when hollow viscus has burst, or removal of tubes in salpingitis. Laparotomy to be considered also in all cases of purulent peritonitis.

4. *Saline purgatives* are of doubtful value in medical cases.

CHRONIC PERITONITIS.

Definition.—Chronic inflammation of the peritoneum.

Varieties.—1. Simple. 2. Tubercular. 3. Cancerous.

Causes of simple form often unknown. *Bright's disease* is a frequent cause.

The *tubercular* form is due to tubercle bacilli. It may occur at *any age*; is common in *women*, and especially common in *negroes*. It frequently extends from the Fallopian tubes, but the tubes are as often affected secondarily.

The *cancerous* form is of course due to cancer and the irritation caused by it.

The *tubercular* is by far the *most common variety*.

Morbid Anatomy.—1. Tubercles may be found distributed over the peritoneum in great numbers (acute miliary form).

2. There may be caseous and ulcerating masses, which may form openings between the different coils of intestines; or the *glands* (mesenteric) may be chiefly involved, enlarged and caseous.

3. There may be great *increase of connective tissue* (fibroid form), causing contraction of the mesentery, coiling and shortening of intestines and folding of omentum to form tumor-like mass.

Symptoms.—1. Often latent.

2. Ascites, from serous exudate.

3. Tympanites, from loss of tone of the muscular coat of the bowel.

4. The *Temperature* may be elevated but is often sub normal ; the reason is unknown.

5. *Pigmentation* of face, &c., is often marked.

Physical Signs.—1. On *inspection* there may be enlargement from (1) ascites, (2) encysted effusion, (3) omental thickening, (4) adhesions of coils of intestines, (5) enlarged mesenteric glands

2. *Palpation* and *percussion* give evidence in the same direction.

Diagnosis.—Tubercular tumors are distinguished from *cancerous* by (1) the existence of tubercular disease elsewhere, (2) possibly sub-normal temperature in tuberculosis, (3) cancer is rare in the young, tuberculosis common.

Tubercular tumors are distinguished from *ovarian tumors* with *great difficulty* by (1) the existence of tuberculosis elsewhere than in the abdominal cavity, (2) the temperative disturbances common in tuberculosis, rare in ovarian tumors.

Complications.—(1) Salpingitis, (2) pleuritis, (3) pericarditis all of which may be due to direct extension of the bacilli, (4) pulmonary phthisis, (5) intestinal tuberculosis.

Prognosis.—Recovery sometimes occurs, but death is most common termination.

Treatment.—1. *Medical.* To sustain strength by food, stimulants, cod liver oil, &c. (See Pulmonary Phthisis.)

2. *Surgical.* *Laparotomy* and *drainage* have given encouraging results.

ASCITES.

Definition and Frequency.—An accumulation of fluid in the peritoneal cavity. It is of frequent occurrence.

Causes.—1. Any *impediment* to the *portal circulation* such as (1) cirrhosis of the liver, (2) thrombosis of the portal vein and pylephlebitis, (3) pressure on the veins by tumors, &c.

2. Diseases of the *heart and lungs* leading to passive hyperæmia.

3. *Chronic peritonitis*, simple or tubercular.

4. *Cancer* of the peritoneum.

5. *Bright's disease*.

Morbid Anatomy.—1. *Enlargement* of the *belly* from the fluid which it contains.

2. *Solden* and *cloudy appearance* of the peritoneum from the action of the liquid.

3. *Displacement of organs* from pressure of fluid.

4. *Various changes* due to *causative* condition. (See Chronic Peritonitis, Cirrhosis of the Liver, &c.)

Symptoms.—1. *Respiratory* symptoms (dyspnœa) are usually most conspicuous; they are due to pressure.

2. *Circulatory.* *Palpitation* of the *heart* occurs from the same cause; œdema of the lower limbs may occur from pressure on the veins; enlargement of the veins on the surface of the abdomen to form collateral circulation.

3. *Digestive* disturbances, dyspepsia and constipation from pressure.

4. *Urinary* symptoms (scantiness) are due usually to lowered blood pressure.

Physical Signs.—1. On *inspection*, enlargement of the belly from fluid.

2. Enlargement of the surface veins on abdomen to form collateral circulation.

On *palpation*, fluctuation from fluid.

On *percussion*, flatness at the sides, resonance in front, as the patient lies on the back, because the fluid sinks to the lowest part; but adhesions may tie the bowels down to the back of the abdomen so as to cause flatness in front (see Chronic Peritonitis).

Diagnosis.—Distinguished from (1) an *ovarian cyst*, by flatness at sides, resonance in front, as a rule, and history of the case; (2) *pregnancy*, by absence of signs of pregnancy; (3) *distended bladder*, by use of the catheter and change of position of fluid when patient changes position; (4) *colloid cancer* of peritoneum by tapping; no fluid flows in colloid cancer.

Prognosis depends on cause.

Treatment.—1. To *remove the cause*, if practicable.

2. To *remove fluid*, by (1) hydrogogue cathartics, (2) diuretics, (3) diaphoretics, (4) aspiration.

Aspiration is indicated whenever respiration is much interfered with.

HYPERÆMIA OF THE LIVER.

Definition Frequency and Synonym.—An excessive amount of blood in the liver. It is probably of common occurrence and is known as "*Congestion of the Liver.*"

Varieties.—1. Active. 2. Passive.

Causes.—A. *Active Hyperæmia.*

1. *Over-eating and drinking*, especially of highly-seasoned food and irritating (alcoholic) liquors, which increase the work to be done by the liver and cause irritant substances to pass into it.

2. *Malaria.* The leucomaines formed in this disease probably pass through the liver and cause irritation.

3. *Heat.* The disease is common in hot climates; the cause is not very evident.

B. *Passive hyperæmia.*

1. Any *obstructive* disease of the heart or lungs which leads to accumulation of blood in the venous system.

2. Any *regurgitant* disease of the heart leading to over-filling of the veins.

3. *Pressure* on the veins leading to the heart.

4. Weakness of the heart's action from any cause, debility, sedentary habits, &c.

Morbid Anatomy.—1. The *size* of the liver is increased, because it contains more blood.

2. The *color* in the passive form especially is darker than normal, because of the amount of venous blood in the organ.

3. In *consistence* the liver is at first softer than normal, because of the serous exudate into it, but subsequently becomes harder from the formation of connective tissue.

4. On *section* it is (1) pigmented from the deposit of the coloring matter of the blood (see Passive Hyperæmia); (2) mottled (nutmeg liver) from the excessive amount of venous blood in the center of the lobules and the lighter color of the cells at the periphery of the lobules; (3) there is an increase of connective tissue around the hepatic veins in the centre of the lobules.

Symptoms.—1. Those due to the *causative disease* such as cardiac troubles, respiratory disturbances, &c. (q. v.)

2. Those due to the *failure of the liver* to perform its functions, such as headache, dullness, stupor, &c., from retention of morbid matters.

3. Those due to the *accompanying conditions*, such as nausea and indigestion from gastric catarrh, slight jaundice from duodenal catarrh, hemorrhoids, &c.

Physical Signs.—1. On *palpation* and *percussion* the liver is found to be *enlarged* from distension with blood and sometimes *tender* from the increased sensibility of the nerves.

Diagnosis based on the presence of the *causes* of the disease and the absence of symptoms of abscess, cancer, &c.

Prognosis.—The *prognosis* depends on the *cause*; in *active* hyperæmia it is usually good; in *passive*, it is usually bad.

Treatment.—To *remove the cause* in cases of

A. Active hyperæmia by unstimulating diet, &c.

B. Passive hyperæmia, by digitalis in failing compensation in heart troubles.

2. To *lessen the engorgement* of the portal system of veins and carry off poisonous substances lying in the stomach and intestines by calomel, salines, the sulphur waters, &c.

3. To *stimulate the liver* (?) by euonymus, podophyllin, muriate of ammonia, &c.

(See Functional Disturbances of the Liver.)

PERI-HEPATITIS.

Definition and Frequency.—An inflammation of Glisson's capsule of rare occurrence.

Causes.—1. Injuries. 2. New growths. 3. Syphilis. 4. Cold possibly.

Morbid Anatomy.—*Size.* The liver is contracted because of the new formation of connective tissue on the surface of the organ and also in the trabeculæ which run in from Glisson's capsule.

Symptoms.—1. *Pain and tenderness* over the liver and slight fever from the inflammation.

2. Sometimes *cough*, from involvement of the diaphragmatic pleura.

3. *Occasionally jaundice and ascites* from compression of the common bile duct and veins by the new formed connective tissue.

Physical Signs.—1. On *palpation* and *percussion* slight enlargement, possibly, *at first*, but contraction afterwards.

2. On *auscultation*. friction sounds are sometimes heard from the rubbing of the surfaces against each other.

Diagnosis.—Distinguished from—1. *Intercostal neuralgia* by the presence of fever and the situation of the pain and tenderness; 2, *pluerisy* by the situation of the pain and friction sounds; 3, *hepatic abscess* by the absence of marked enlargement of the liver and of sweats and repeated chills which *sometimes* occur in abscess.

Prognosis.—The *prognosis* is generally good.

Treatment.—To *relieve pain* and *subdue inflammation* by rest, poultices, counter-irritants. In the case of syphilis, iodide of potassium should be employed.

INTERSTITIAL HEPATITIS.

Definition, Synonym and Frequency.—*Interstitial Hepatitis*, or *Cirrhosis of the Liver*, is a disease characterized by a great increase in the amount of connective tissue in the liver and atrophy of its parenchyma. It usually runs a chronic course.

Causes.—1. *Alcohol* is by far the most common cause; it is uncertain whether it causes atrophy of the cells *first*, and then an increase of connective tissue or whether the increase of connective tissue is the primary change, and the atrophy of the cells is due to pressure.

2. *Highly-seasoned food and malarial poisoning* are rare causes; both act by setting up an irritation in the liver.

3. *Syphilis* unquestionably causes one form of cirrhosis.

Morbid Anatomy.—1. The *size* of the liver is at first greater than normal, because of the exudate of cells and fluid into it; but later it is small, owing to the contraction of the new-formed connective tissue.

2. *Color, consistence and appearance.* Its color is lighter (hence its name, cirrhosis), its consistence firmer, and its surface irregular ("hob-nailed"), from the increase of connective tissue.

3. On *section*, in (1) the *early* stages the liver is filled with leucocytes; (2) in the *late* stages there is a great increase of connective tissue formed from the leucocytes and an atrophy of the parenchymatous tissue.

4. There is *passive hyperæmia* and its results in the organs connected with the portal system, because the portal vessels are compressed by the new formed connective tissue.

Symptoms.—1. *Digestive.* Anorexia, nausea and furred tongue, from the failure of the liver to remove waste products, and from the accompanying passive hyperæmia and congestion of the stomach and bowels.

Constipation is usual from the diminution in the amount of the bile formed; but *diarrhœa* may occur from the fermentation of food and the catarrhal enteritis caused thereby.

Jaundice sometimes occurs from pressure of the connective tissue on the bile ducts.

Ascites almost invariably occurs from the compression of the portal vessels by the connective tissue and the consequent passive hyperæmia of the portal system and the transudation therefrom.

2. *Respiratory*. Dyspnœa occurs from the distension of the abdomen with ascitic fluid.

3. *Circulatory*. Gastric and intestinal hemorrhage from the passive hyperæmia and enlargement of abdominal veins from collateral circulation.

4. *Nervous*. Apathy, depression, delirium, convulsions and so called "cholæmia," from the retention of substances which should have been excreted or acted upon by the liver.

5. *Urinary*. Decrease in the amount of urine from lowered blood pressure in the arterial system; diminution in the amount of urea from destruction of hepatic parenchyma; increase of urates and presence sometimes of leucine and tyrosine from the same cause.

Physical Signs.—1. On *inspection*, enlarged veins on abdomen and caput medusæ around umbilicus (collateral circulation), distension of abdomen from ascitic fluid.

2. On *palpation*, fluctuation, from the presence of fluid, enlarged spleen, from passive hyperæmia; sometimes irregular surface of the liver can be felt.

3. On *percussion*, enlarged spleen and ascites (q. v.).

Diagnosis.—1. Distinguished from *fatty liver*, *waxy liver* and *cancer* of liver by the absence of enlargement.

2. From *chronic peritonitis*, by the history of alcoholic excesses in cirrhosis.

Prognosis and Duration.—The prognosis is very bad, but not absolutely hopeless; the duration is from a few months to several years.

Treatment.—To *remove the cause*; to forbid the use of alcohol, spices and highly-seasoned food; the most suitable diet is milk.

2. To *relieve congestion* and pressure symptoms by hydragogue cathartics, diuretics, diaphoretics and tapping.

Tapping, in some cases, if *repeated often*, will lead to arrest of the disease, or, at least, the arrest of the dangerous symptoms.

3. To *relieve symptoms* as they arise.

ABSCESS OF THE LIVER.

Definition, Synonym and Frequency. An inflammation of the liver leading to the formation of one or more collections of pus. It is sometimes called *suppurative hepatitis*, and is of comparatively rare occurrence.

Causes.—The lodgment of septic emboli (*pyæmia*).

2. *Ulcerative disease* of the bowels, such as dysentery, in which infective emboli may be carried to the liver through the portal system.

3. *Heat*; the disease is most common in hot countries, probably because the conditions for germ growth are favorable there.

Often no cause can be discovered.

Morbid Anatomy.—1. *Number*. They may be single or multiple.

2. The *size* varies from a minute point to a sack which holds five or six pints.

3. Condition of the *walls* and *surrounding tissue*. The walls may be irregular and caseous, or in older cases a pyogenic membrane may be present. The surrounding tissue is inflamed.

4. *Characters of the pus*. It may be liquid and yellow, or brownish in color; it may be cheesy.

Symptoms.—*Often there are none*. Usually, however, there are—

1. *Digestive*. Nausea and indigestion from failure of the liver to excrete waste matters, occasionally jaundice from pressure on a bile duct.

2. *Respiratory*. Dyspnœa, if a large abscess presses upwards on the diaphragm.

3. *Urinary*. Absence or great diminution of urea and the presence of leucine and tyrosine from the failure of the liver to burn off the nitrogenous waste.

4. *Nervous*. Depression from retention of excrementitious matters; pain often, but not always, from localized peritonitis; hic-cough.

5. Occasionally chills, fever and sweats when pus is forming; but pus often forms insidiously.

Physical Signs.—1. On *inspection*, there may be some prominence over the liver.

2. On *palpation*, a swelling can usually be felt and frequently fluctuation.

3. On *percussion*, the outlines of the swelling can be determined.

Diagnosis.—1. From *cancer of the liver* it is distinguished by the history of the case and especially by *aspiration*.

2. From *enlarged gall bladder* by the situation and shape of the latter.

3. From *sub-diaphragmatic abscess* (localized peritonitis with supuration) by the history of the case; the diagnosis here is extremely difficult.

Prognosis.—Always serious, but recovery may occur.

Duration from a few weeks to several years.

Results.—1. *Death* from (1) peritonitis (from bursting); (2) exhaustion.

Bursting may occur into (1) peritoneal cavity; (2) plueral cavity or bronchus; (3) intestines; (4) pelvis of the kidney, or (5) through the abdominal wall.

2. *Recovery* may occur from (1) bursting in favorable direction; (2) absorption; (3) calcification.

Treatment.—1. To *relieve pain* by opiates, phenacetine, hot poultices, &c.

2. To *sustain strength* by nutritious food and stimulants (when absolutely necessary.)

3. To *evacuate* the pus by

(1) *aspiration*, which is but little dangerous, or

(2) *free incision and drainage*, in which the liver should be fastened to the abdominal wall before the abscess is opened.

Union may be obtained between the liver and abdominal walls by either (1) cutting down on the liver and stuffing the wound with carbolized gauze till adhesions have formed and then cutting into the liver, or (2) cutting down upon the liver, *sewing* it to the abdominal wall and opening the abscess at once.

ACUTE YELLOW ATROPHY OF THE LIVER.

Definition and Frequency.—A *very rare* disease characterized *anatomically* by atrophy of the liver and change in its color, and *clinically* by severe nervous symptoms which usually terminate in death. The disease is *probably* an *acute infectious* one, but it is not certain.

Causes.—*Pregnant women* between the ages of fifteen and thirty-five are most liable to the disease.

Morbid Anatomy.—1. The *body* is yellow from absorbed bile.

2. The *liver* is soft, small, shrivelled and reddish-yellow in color; the cells are fatty or granular and many have disappeared entirely.

The *spleen* is enlarged; there may be fatty degeneration of the heart and kidneys.

Symptoms.—1. *Digestive.* Anorexia, nausea and vomiting, and jaundice.

2. *Temperature* is elevated— 101° to 103° .

3. The *pulse* is rapid and feeble.

4. The *urine* contains *very little* or no urea, but a large amount of leucine and tyrosine.

5. *Nervous*. Headache, stupor, delirium and often convulsions and coma, probably from the absorption of leucomaines.

Physical Signs.—On *palpation and percussion*, the liver is found to be greatly reduced in size.

Diagnosis based on the symptoms and course of the disease.

Prognosis bad.

Treatment.—Symptomatic.

PYLEPHLEBITIS.

Definition and Varieties.—An inflammation of the portal vein or its branches, which may be suppurative or adhesive.

Causes.—1. *Suppurative* form is chiefly extension from veins of the intestines, as in appendicitis or lodgment of an embolus. (See Abscess.)

2. *Adhesive* form. Cirrhosis and hepatitis, which compress the veins and interfere with their nutrition (See Thrombus in General Pathology.)

Morbid Anatomy.—1. In *septic* form, suppuration and abscess.

2. In *adhesive* form *thickening of the coats* of the vessel from inflammation and then the formation of thrombi.

3. *Changes* due to *obstruction* of the vein; passive hyperæmia of the abdominal organs; splenic enlargement and ascites.

Symptoms.—1. *Rapid ascites* from sudden stopping of vein.

2. *Enlarged spleen*.

3. *No jaundice*.

Diagnosis from *cirrhosis* based on *sudden* occurrence of the ascites and different history as to cause.

Prognosis bad.

Treatment.—Palliative and symptomatic.

AMYLOID DEGENERATION OF THE LIVER.

Synonyms and Frequency.—A rare disease of the liver, called also "*lardaceous*" and "*waxy*" degeneration.

Causes.—1. *Age and sex*. Men between the ages of 20 and 50 are most liable to it.

2. *Syphilis*.

3. *Suppuration*, especially when bones are involved.

Morbid Anatomy.—1. The *size* is much greater than normal.

2. The *consistence* is firm, but it is brittle.

3. The *appearance* is smooth, and on microscopic examination the outlines of the cells cannot be found. Tincture of iodine poured over the cut surface causes a mahogany color.

4. The *kidneys* and bowels are usually involved also.

Symptoms.—Not marked; no jaundice; no pain.

1. *Weight* and *heaviness* in abdomen from enlargement of the liver.

2. *Diarrhœa*, when the bowels are involved.

3. *Albuminuria*, when the kidney's are involved. (See Bright's.)

Physical Signs.—On *palpation* and *percussion*, the liver is found greatly enlarged.

Diagnosis.—Distinguished from fatty liver by accompanying albuminuria and the presence of the causes.

Prognosis.—Unfavorable; duration often several years.

Treatment.—1. Removal of cause.

2. Antisymphilitics, if syphilis is present.

3. Alkalies and iodide of iron.

FATTY DEGENERATION OF THE LIVER.

Forms.—1. Fatty infiltration.

2. Fatty metamorphosis.

Causes.—(See *Fatty Degeneration* in General Pathology).

Morbid Anatomy.—1. The *size* of the liver is greatly increased.

2. The *color* is yellower than normal, and the *consistence* is softer.

3. On *section*, the cells are distended with fat.

Symptoms.—Not marked; no jaundice; no ascites; no pain.

1. *Weight* and *fullness*, from enlargement of the liver.

2. Possibly *gastric catarrh*.

3. *Greasy* condition of the skin.

Physical Signs.—Those of enlargement of the liver.

Diagnosis.—(See *Waxy Degeneration*).

Prognosis.—Not serious; duration indefinite.

Treatment.—1. *Dietetic*. The avoidance of saccharine and starchy food; skimmed milk and lean meat are useful.

2. *Active, out-of-door* life, to increase oxidation.

3. *Cold* climate, for the same reason.

CANCER OF THE LIVER.

Varieties.—1. Scirrhus. 2. Medullary.

(1) primary; and

(2) secondary to cancer elsewhere.

Causes.—1. *Age.* After forty usually; it *may* occur earlier.

2. It is often *secondary* to cancer elsewhere, as in the stomach.

Morbid Anatomy.—1. The *size* of the liver is greatly increased, usually; the individual nodules of cancer vary from a very small size to the size of a hen's egg.

2. *Appearances.* There are usually white, depressed spots on the surface and in the interior of the liver, from contraction of the connective tissue stroma of the cancer nodules.

Symptoms.—Often obscure in the beginning.

1. *Pain, emaciation and cachexia*, as in cancer elsewhere.

2. *Jaundice*, occasionally, when a cancer nodule presses on the bile ducts.

3. *Ascites*, sometimes, from the compression of the portal vein by the cancer.

Physical Signs.—On *palpation* and *percussion*, there is found an enlargement of the liver, usually hard and nodular (in scirrhus), but sometimes soft (in medullary form).

Diagnosis.—Distinguished from *abscess* by the severe pain, cachexia and emaciation, but especially by the results of aspiration.

Prognosis.—Always unfavorable; duration usually about a year.

Treatment.—Opiates, phenacetine, &c., to relieve pain.

HYDATIDS OF THE LIVER.

Definition.—A cyst in the liver caused by the *tænia echinococcus*.

Causes.—The entrance into the alimentary canal—from whence it passes to the liver—of the *tænia echinococcus*. It is taken with water contaminated with the excrement of dogs.

Morbid Anatomy.—1. The *size* of the cyst varies from a minute point to the size of the head.

2. The *walls* consist of several layers of homogeneous substance and an inner layer of cells from which daughter cysts may grow. Around the cyst is a wall of connective tissue. The fluid contains no albumin, but little hooklets from around the heads of the *tæniæ*.

The cyst may (1) burst; (2) suppurate; (3) undergo calcification.

Symptoms.—Not characteristic; due to pressure on neighboring organs.

Physical Signs.—On *palpation* and *percussion* there is enlargement and sometimes the hydatid thrill.

Diagnosis based on aspiration and character of aspirated fluid.

Prognosis and Duration.—Hydatids of the liver may last for years and are harmless except for complications.

Treatment.—1. *Prophylactic.* Consisting especially in the purification of the drinking water.

2. *Remedial.*

(1) *aspiration* and injection with tincture of iodine successful in about 60 per cent. of cases; but often fails to kill the hydatids;

(2) *free incision* and drainage, as in abscess of the liver, usually very efficacious;

(3) *excision* of the cyst and part of the liver has been practised two or three times with success, but is still on trial.

FUNCTIONAL DERANGEMENTS OF THE LIVER

Causes.—1. Anything which interferes with the proper functions of the liver, such as (1) cardiac diseases; (2) malaria; (3) specific fevers; (4) sedentary habits.

2. Overtaxing of the liver with improper quality or inordinate quantity of food.

Symptoms.—1. Nausea, vomiting, headache, &c., from defective elimination of waste products.

2. Slight jaundice, constipation and flatulence, from diminished discharge of bile.

Treatment.—1. *Dietetic.* Simple food, as skimmed milk, fresh meat, &c.

2. *Blue mass and calomel, salines, &c.*, to wash out the bowels and possibly prevent formation of ptomaines.

JAUNDICE

Definition and Synonym.—Jaundice, or *icterus*, is the yellow color of the skin and other tissues, produced by the circulation of bile or biliary coloring matter in the blood.

General Causes.—It is usually due to some *obstruction to the outflow of bile*, and such obstruction may be

1. Plugging of the common bile duct by tough mucus, swelling of the mucous membrane or gall stones.
2. Pressure on the duct from without by cancerous masses, connective tissue (in cirrhosis or perihepatitis), &c.

Sometimes, however, there is no obstruction of the duct which can be discovered. In these cases the jaundice is called *hæmatogenous*. In *hæmatogenous* jaundice it was formerly thought that the liver failed to remove bile from the blood; this view has, of course, been abandoned, but no explanation of cases of hæmatogenous jaundice has yet been found.

It is probably due to a rapid destruction of red blood corpuscles.

CATARRHAL JAUNDICE.

Definition.—Jaundice due to catarrhal inflammation of the bile ducts. It is a common affection.

Causes.—1. *Gastro duodenal catarrh* often causes it by *extension* to the bile ducts and consequent swelling of the mucous membrane, and also by the formation of tough mucus.

2. *Structural diseases of the liver*, which cause hyperæmia from obstruction of the veins.

3. Any *other impediments* to the *return circulation*, such as certain heart or lung troubles.

4. *Syphilis*, which probably causes catarrhal inflammation by the pressure of a gumma on a vein.

5. *Pyæmia*, the action of which is obscure.

6. *Gall stones*, which set up irritation mechanically.

7. Possibly *exposure to cold*.

Morbid Anatomy.—1. The *skin and other tissues* are yellow from deposition of biliary coloring matter.

2. The *bile ducts* are swollen, red, and covered with muco-pus.

3. The *liver* is enlarged and yellow, and the *gall bladder* is enlarged and full of bile.

Symptoms.—1. Yellowness of the skin, sclerotic, &c., from absorption of biliary coloring matter into the blood and its subsequent deposit in the tissues.

2. *Nausea and vomiting*, from the retention in the blood of waste products or leucomaines. *Clay-colored stools, constipation and flatulence*, sometimes *diarrhœa*, from the absence of bile from the intestines.

3. The *urine is brown*, and on addition of nitric acid to biliary

urine in a saucer, there is a play of colors where the two fluids come together.

4. The *pulse* is slower than normal, probably from the action of retained matters on the inhibitory apparatus.

5. The *temperature* is normal or possibly below normal.

6. The *nervous* symptoms are headache, apathy, vertigo, &c, and are due to the absorption of leucomaines or the retention of waste products.

Violent itching is usual, and is probably due to the irritation of the terminal filaments of the nerves by the bile.

Physical Signs.—1. On *inspection*, jaundice.

1. On *palpation* and *percussion*, sometimes there is slight enlargement from the accumulation of bile, and also slight tenderness.

Diagnosis.—1. From *cancer*, by the absence of pain and cachexia.

2. From *gall stones*, by the absence of colic.

Prognosis.—In simple *catarrhal* jaundice uniformly good, unless there is obstruction from gall stones, &c.

Treatment.—1. *Diet.* Fats should be avoided; skimmed milk and lean meat are suitable.

2. Phosphate of soda, alkalies and alkaline waters, which seem to lessen the tenacity of the mucus, and also to allay the gastro-duodenitis.

3. *Pilocarpine*, to relieve itching.

GALL STONES.

Definition.—Calculi found in the gall bladder, or rarely in the biliary ducts in the liver, composed chiefly of cholesterine.

Causes.—1. *Age and sex.* Most common in women between twenty-five and forty.

2. *Sedentary habits* seem to predispose to their formation.

Morbid Anatomy.—1. *Characteristics of the calculi.* Usually multiple in *number*; the *size* varies from sand to the size of a hen's egg; they usually show *facets* from pressure against each other; they are brown in *color*, and composed chiefly of *cholesterine* with some *pigment*.

2. *Condition* of the gall bladder. As a rule, the gall bladder is in a state of chronic inflammation.

3. There may be *fistule* between the gall bladder and the stomach or bowels.

Symptoms.—None, unless a stone lodges in the common bile duct; then *gall stone colic* occurs, characterized by violent pain under the ribs on the right side; the pain radiates to the back, is very intense, and often causes extreme prostration. It is usually followed by jaundice.

Diagnosis.—Gall stone colic is distinguished from *simple colic* by the absence of flatulence and the seat of the pain; from *renal colic* by the seat of the pain and the presence (sometimes) of blood in the urine in the latter disease.

Prognosis.—The *prognosis* is generally good, but a stone may lodge in the gall duct and cause *dropsy of the gall bladder*, or may even cause obstruction of the bowels.

Treatment—1. To *relieve pain* and *relax spasm* by opiates, chloroform, hot baths, phenacetine, &c.

2. To *prevent the formation* of gall stones by choleate of soda, alkalies, and alkaline waters.

3. To *remove the stone* by cholecystotomy, or the gall bladder by cholecystectomy.

DROPSY OF THE GALL BLADDER.

Causes.—1. *Catarrh* of the *gall duct*, causing swelling and obstruction.

2. *Gall stones* stopping up the canal.

3. *Outside pressure* on the gall duct, from cancer, &c.

Morbid Anatomy.—1. The *size* of the gall bladder is greatly increased.

2. The *wall* is thickened.

3. The *contents* are at first bile, but afterwards the bile is absorbed and a mucous secretion takes its place.

Symptom.—None characteristic.

Physical Signs.—On *palpation* and *percussion*, a pear-shaped, fluctuating tumor underneath the ribs on the right side.

Diagnosis.—1. From *abscess*, by the seat and size of the enlargement and the fluid drawn by the aspirator.

2. From *cancer*, by the absence of pain and cachexia.

Prognosis.—Serious, because of the difficulty usually in removing the cause.

Treatment.—1. To *relieve the catarrh* of the bile ducts.

2. To *remove the gall stones*.

3. To *empty the gall bladder* by aspiration,

DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS.

Frequency.—The disease is probably very rare.

Causes.—*Gastro-duodenitis* has been the cause usually in those cases which have been carefully studied.

Morbid Anatomy.—It may occur in one of three forms—(1) suppurative; (2) hemorrhagic; (3) gangrenous.

Symptoms.—The *symptoms* come on suddenly, and are those of peritonitis limited to the pancreatic region.

Prognosis.—The *prognosis* is grave in all cases.

Treatment—is limited to sustaining strength and relieving pain. At a later stage there is a possibility that good may be accomplished by surgical interference.

Nothing definite is known with respect to *chronic pancreatitis*.

Pancreatic cysts, due to the obstruction of the pancreatic duct, have been frequently observed, and have occasionally been cured by surgical means.

DISEASES OF THE SPLEEN.

Affections of the spleen are nearly always secondary, and are of little practical importance, except in the diagnosis of the acute infectious diseases.

Congestion of the spleen occurs in all affections leading to *passive hyperæmia of the abdominal viscera*, such as cirrhosis of the liver and certain cardiac diseases.

It occurs also in the *acute infectious diseases*, probably as a result of the circulation of leucomaines through it.

Splenitis is rare; it may lead to an increase in the connective tissue of the organ or to abscess formation in cases of septic infarction.

Waxy degeneration of the spleen sometimes occurs, but is in itself of no practical significance.

Enlargement of the spleen, which is often permanent, is especially common after malarial affections. It gives rise to no special symptoms, but is readily detected on palpation and percussion.

The anti-malarial remedies will frequently cause the disappearance of the splenic enlargement.

CHAPTER V.

DISEASES OF THE HEART.

PERICARDITIS.

Definition and Frequency.—Inflammation of the pericardium. It is quite frequent.

Causes.—It is *rarely primary*.

1. *Acute rheumatism* is the most common cause. The pericarditis is probably due to the morbid matter circulating in the blood and lymph.

2. The *acute infectious* diseases, especially septicæmia and the exanthemata, cause it through the action of leucomaines.

3. *Bright's disease* (nephritis) probably acts in consequence of the retention of irritating waste products.

4. *Tuberculosis* is a common cause.

5. *Injuries* may cause it, but rarely do so.

Morbid Anatomy.—1. The changes in the pericardium consist in *cloudiness* of the endothelial cells, and later, *roughness* from fibrinous exudate; redness is, of course, present.

2. The *muscular tissue of the heart* is more or less infiltrated with serum and cells, and its fibres undergo cloudy swelling, and possibly fatty degeneration later on; or connective tissue may form in spots.

3. The *exudate* may be serous, sero-fibrinous, fibrinous, purulent or hemorrhagic; usually it is fibrinous at first, and serum is subsequently added.

4. *Results.* The pericardium may be *shaggy* from the rubbing together of the fibrinous exudate on the opposing surfaces of the membrane; or there may be *adhesions* more or less extensive between the costal and cardiac pericardium; or there may be *calcification* of the exudate (so-called ossification of the heart).

Absorption is possible, but very rare.

Symptoms.—The symptoms are often latent or overshadowed by the causative disease.

1. *Cardiac.* Pain and *constriction* in the præcordial region from the pressure of the exudate on the nerve filaments of the pericardium and *palpitation* from the efforts of the heart muscle to neutralize the effects of pressure of the effusion.

The *pulse* is quick, jerky, and when there is much effusion weak, because the pressure of the effusion on the heart interferes with the filling of its cavities, and hence only a small quantity of blood is thrown into the vessels by each systole.

2. *Respiratory.* Dyspnœa is present when there is much effusion, because very little blood is forced into the lungs to be oxygenated.

3. The *temperature* varies; it is usually elevated somewhat— 101° to 103° —but in septic and some rheumatic cases may be higher.

Physical Signs.—1. On *inspection*, there may be cyanosis, from want of oxygenation of blood; the præcordial region may be distended with effusion.

2. On *palpation*, the heart's impulse is often feeble, from the serous fluid surrounding the organ. The rubbing together of the pericardial surfaces may be felt.

3. On *percussion*, the area of cardiac dullness may be greatly increased by the serous exudate.

4. On *auscultation*, friction sounds may be heard, which are due to the rubbing of the roughened pericardial surfaces against each other; but these friction sounds may be prevented by (1) serous effusion, (2) adhesions.

Diagnosis.—1. From *endocarditis* it is distinguished by the more superficial character and greater roughness of the sounds. Pericardial sounds do not extend far.

2. From *pleurisy* by the seat of the sounds and the fact that they continue while the patient holds his breath.

3. From *hypertrophy* by the fact that the apex beat in pleural effusion is not at the limit of the dullness; the history of the case also is different.

Prognosis.—The prognosis is usually favorable as to life, except in (1) septicæmia, (2) nephritis.

The *duration* is from one to three weeks.

Results.—More or less *adhesion* of the pericardial surfaces always occurs. *Dilatation* of the heart may occur from softening of the cardiac walls and intra-cardiac pressure.

Treatment.—1. To *check inflammation* by (1) absolute quiet; (2) aconite; (3) veratrum, and in the late stages by blisters; digitalis, if the heart's action is feeble.

2. To *relieve the causative diseases* (q. v.)

3. To *relieve pain*, by opiates, which should be given cautiously, and by counter-irritants or hot applications.

4. To *sustain strength*, by suitable diet; stimulants to be avoided, if possible.

5. To *promote absorption* by iodides, iron, stimulants, blisters, &c.

6. To *remove serous or purulent exudate* by—

(1) *aspiration*, in cases of serous exudate which is so large as to endanger life;

(2) *free incision and drainage* (very rarely) if the exudate is purulent.

HYDRO-PERICARDIUM. HÆMO-PERICARDIUM. PNEUMO-PERICARDIUM.

Hydro-pericardium is due to passive hyperæmia, &c., and should receive the same treatment as the other forms of dropsy.

Hæmo-pericardium is a collection of blood in the pericardial cavity; it may be due to the breaking of adhesions or to hemorrhagic disease, or to inflammation. The *treatment* is chiefly palliative.

Pneumo-pericardium is the presence of air or gas in the pericardium. It may occur from injury or ulceration.

ENDO-CARDITIS.

Definition and Frequency.—Inflammation of the lining membrane, of frequent occurrence.

Varieties.—1. Acute exudative; 2, ulcerative.

Causes.—Rarely idiopathic or primary.

1. The same as those of pericarditis (q. v.).
2. Chona; it is not known why it causes endo-carditis.

Morbid Anatomy.—A. Exudative. 1. *Seat.* The *left side* of the heart is nearly always involved because of strain on it; it occurs in patches, and the *valves* are most commonly affected.

2. *Structural changes.* *Papillary excrescences*, composed of leucocytes, form usually on the edges of the valves; the endothelial cells over these undergo cloudy swelling and degeneration, and *fibrin* is deposited on the excrescences. The edges of the valves may adhere to each other, causing narrowing of the valvular opening (*obstruction*), or the valves may be crumpled from contraction of new connective tissue, or the chordæ tendinæ may be shortened for the same reason; both conditions cause *insufficiency* of the valves.

B. *Ulceration.*—1. *Seat.* Usually on *valves* of *left side*.

2. *Micrococci* are present and lead to

3. *Ulcerative*, which may cause a piece of the valve to be broken off, or may cause

4. *Valvular aneurism*, from pressure of the blood on the valve, thinned by the ulceration.

5. Pyæmic abscess in other organs are common from septic emboli.

Symptoms.—The subjective symptoms are usually very ill-defined.

1. The *pulse* is quick and jerky, but not full, because the heart is more irritable than normal, and so contracts before its cavities become fully distended with blood. In the ulcerative form it is rapid and feeble.

2. The *temperature* is elevated, probably from the absorption of

leucomaines, in the exudative cases; in these cases it is rarely over 103° .

In the *ulcerative* form, the temperature often reaches 106° , and may be preceded by a chill and followed by sweating; it is due to absorption of septic matter.

3. The *respiration* is quickened in consequence of the increased rapidity of circulation and the elevated temperature (see Fever).

4. *Typhoid symptoms* are common in the ulcerative form, and also *dyspnea* and *cyanosis*; these latter are probably due to the lodgment of septic emboli in the pulmonary vessels.

Physical Signs.—1. On *inspection* and palpation the cardiac impulse is marked, from irritability of the heart.

2. On *percussion*, no information of importance can be obtained.

3. On *auscultation*, in fresh cases and first attacks, a soft, blowing sound is heard, commonly at the apex, from the passage of the blood over the excrescences in the valves, or from the formation of "whirls" in the blood by obstruction or regurgitation.

Diagnosis.—1. From *pericarditis* by softness and greater distance of the sound.

2. From *functional* cardiac murmurs by the history of the case (*usually rheumatic*), and by the *situation* of the murmurs, functional being at the base and those of endo-carditis usually at the apex.

Prognosis.—The prognosis, as to *life*, usually good in the exudative form; almost hopeless in the ulcerative.

The exudative form usually leaves crippled valves.

Results.—1. *Calcareous degeneration* of vegetations (excrescences) on the valves.

2. *Embolism*, which is *simple* in exudative form and septic, leading to pyæmia, in ulcerative.

3. *Valvular disease*. (1) Adhesions of the edges; (2) crumpling; (3) calcareous degeneration, in 25 per cent. of the cases.

Obstruction at a valve may be caused by thickening or adhesions.

Insufficiency may be caused by (1) crumpling of valves; (2) contraction of chordæ tendinæ.

Treatment.—1. To *relieve* the *causative condition*, care being taken not to cause prostration by excessive use of salicylates.

2. To *allay irritability* of the heart by absolute quiet, aconite or veratrum, if pulse is strong, digitalis, if pulse is weak.

3. To *relieve pain* by counter-irritants, hot applications and morphia, if absolutely necessary (pain is rarely severe).

4. To *sustain strength* by nutritious food and stimulants. In the *exudative* form stimulants should be used guardedly; in the *ulcerative* form, freely.

VALVULAR DISEASES OF THE HEART.

Causes of Cardiac Murmurs : 1. *Obstruction* to the flow of blood at the cardiac orifices.

2. *Regurgitation* of blood at the cardiac orifices.

3. Change in the *quality* of the blood.

4. *Spasmodic* contraction of the chordæ tendinæ.

Time and Nature, Seat, and Area of Diffusion of Valvular Murmurs :—

Valve.	Nature of Affection.	Time of Occurrence.	Seat of Greatest Distinctness.	Area of Diffusion.
1. Mitral.	Obstruction.	Presystolic.	Apex.	Towards the left.
2. " "	Regurgitation.	Systolic.	Apex.	Towards the left and behind at lower angle of scapula.
3. Aortic.	Obstruction.	Systolic.	Second right intercostal space.	Along the large arteries.
4. " "	Regurgitation.	Diastolic.	" "	Over the sternum.
5. Tricuspid.	Obstruction.	Presystolic.	Ensiform cartilage.	Unknown, disease very rare.
6. " "	Regurgitation.	Systolic.	" "	Slight ; not heard to the left or above third rib.

Pulmonary valvular sounds are extremely rare, except *accentuation* of the second sound from obstruction to the flow of blood in the lungs ; the sounds are heard most distinctly at the second left intercostal space, close to the sternum.

Exocordial murmurs are (1) pericardial ; (2) cardio-respiratory ; the latter occur at the apex, are systolic in time and due to the forcing of air out of a bronchus or air cells by the cardiac pulsation.

Causes of Valvular Diseases :—1. *Endo-carditis* is by far the most common cause of valvular disease (see "Results" of Endo-carditis).

2. *Age*. Disease of *pulmonary* valves usually congenital, because right side of the heart does more work than the left in fœtal life.

Mitral disease most common in the *young*, because rheumatism is more common in early life.

Aortic disease is common in later life, because it may arise from extension of atheroma from the aorta to the aortic valves.

3. *Occupation*. Laborious occupation may cause aortic disease from increased tension in the vessels and consequent strain on the aortic valves.

4. *Obstruction* to the flow of blood in the *systemic arteries* may cause overfilling of left ventricle and consequent regurgitation of the mitral valve.

5. *Emphysema* and other obstructions to the flow of blood in the lungs lead to distension of the *right* ventricle and tricuspid regurgitation.

Morbid Anatomy.—1. The *size* of the heart is increased in nearly all valvular affections from *compensatory hypertrophy*.

2. The valves (see "Results" of Endo-carditis) may (1) be crumpled; (2) be adherent at their edges; (3) have calcareous nodules on them; (4) be torn so as to have a strip hanging loose in the cavity; (5) be prevented from closing by contraction of chordæ tendinæ.

3. Changes in other organs occur when compensation fails and consist in passive hyperæmia of the lungs, liver, kidneys, bowels, spleen, &c.

AORTIC OBSTRUCTION, OR STENOSIS.

(*Narrowing of the Aortic Opening.*)

Symptoms.—The *symptoms* are not well-marked, indeed, are scarcely noticeable when compensation is good.

The *pulse* is small and slow, because the narrow aortic opening prevents the ventricle from throwing much blood into the arteries and it enters slowly.

Results.—1. *Hypertrophy* of the *left* ventricle, because it has to do more work to force the blood through the narrow aortic orifice.

2. *Secondary insufficiency* of the mitral valve from failing compensation and dilatation.

3. *Embolism* may occur from the breaking off a little piece of a calcareous mass and its lodgment in an artery.

Physical Signs.—1. On *inspection* the impulse of the heart is found to be increased in force and the apex beat is farther towards the *left* than normal, because of enlargement of the left ventricle.

2. On *palpation* the same information may be obtained.

3. On *percussion* the area of cardiac dullness is increased because of the hypertrophy of the left ventricle.

4. On *auscultation* a systolic murmur heard most distinctly at the second right costal interspace; *systolic* because the contraction of the ventricle is forcing the blood through a narrowed orifice, and heard most distinctly at the second right costal interspace, because the arch of the aorta is nearest to the surface at that point. The *area of diffusion* is in the direction of the large arteries, because the blood current sweeps the sound with it.

The sound is usually loud, because the ventricle is strong.

Diagnosis.—1. From *mitral regurgitation* by (1) the point of greatest distinctness, and (2) the area of diffusion.

2. From *tricuspid regurgitation* in the same way.

3. From an *aneurism* by the absence of dullness and the seat of reatest distinctness of the sound.

AORTIC INSUFFICIENCY.

(Aortic regurgitation.)

Symptoms.—Very slight when compensation is good. Later there are—

1. *Dyspnoea* from dilation of left auriculo-ventricular opening and engorgement of the lungs with blood.
2. *Syncope, headache, vertigo, &c.*, from insufficient supply of blood to the brain.
3. *Cardiac* and *præcordial* pain from loss of balance between heart power and vascular tension.

Results.—1. *Slight dilatation* from reflux of blood from the aorta during diastole.

2. *Compensatory hypertrophy* of left ventricle from increased work required of it to force out the excessive quantity of blood.
3. *Failing compensation* and *dilatation* from the giving way of the heart muscle.

Physical Signs.—1. On *inspection*, evidences of cardiac hypertrophy.

2. On *palpation*, evidences of cardiac hypertrophy and “water hammer” pulse in the arteries, because a large quantity of blood is thrown into the vessels with each ventricular systole; but the insufficiency of the aortic valves permits a reflux, and the arterial pressure is suddenly lowered.

3. On *percussion*, the evidences of cardiac hypertrophy, especially of the left side.

4. On *auscultation*, a *diastolic* murmur heard most distinctly at aortic spot, and with slight diffusion, because the current is back towards the ventricle; area of diffusion over the sternum.

Diagnosis.—1. From *mitral obstruction*, by set of the greatest distinctness of the murmur, character of the pulse and hypertrophy of the left ventricle.

2. From a *pericardial friction sound*, by the more blowing and more distant character of the sound.

MITRAL OBSTRUCTION, OR STENOSIS.

(Narrowing of left auriculo-ventricular opening.)

Symptoms.—Not marked unless narrowing is decided; then there are—

1. *Pulmonary.* *Dyspnoea*, and sometimes spitting of blood from passive hyperæmia of the lungs, induced by obstruction at mitral valve.

2. *Circulatory.* The *pulse* is very feeble, because little blood enters the left ventricle; often it is irregular for the same reason.

Results.—1. *Small left ventricle*, because it does not receive its normal supply of blood, consequently has too little work to do.

2. *Large left auricle*, because the blood gets out of it with difficulty, and it is constantly over-distended.

3. *Passive hyperemia of the lung*, from obstruction to the flow of blood at the mitral valve.

4. *Hypertrophy* of right ventricle, because it has to do more work in forcing the blood onwards.

5. *Dilation* of right ventricle (failing compensation) and *tricuspid regurgitation*, leading to general passive hyperæmia and its results.

Physical Signs.—1. On *inspection* the apex beat is but little displaced (if at all) because the left ventricle is not enlarged. There is often pulsation of the *right* ventricle seen below the ensiform cartilage.

2. On *palpation* weak apex beat for obvious reasons and often a *purring thrill* due to vibrations of the thin wall of the auricle. Requisites for a purring thrill are (1) increased propelling force; (2) over distension of cavity with thin walls; (3) disease at the orifice.

3. On *percussion* increased cardiac dullness, especially towards the right, because the right ventricle is chiefly hypertrophied.

4. On *auscultation*, *presystolic* murmur heard most distinctly at the apex and often *blubbery* in character, from the vibration of the valves. Accentuation of second sound at pulmonary valves, from increased tension in *pulmonary* vessels.

Diagnosis.—The *diagnosis* is based on the *seat*, *time* and *character* of the murmur and the *purring thrill*.

MITRAL REGURGITATION.

(*Mitral insufficiency*).

Symptoms.—1. *Cardiac*. The pulse is full and strong so long as compensation lasts, because the left ventricle is enlarged; it is often irregular.

2. *Pulmonary*, are like those of mitral stenosis.

Results.—1. *Hypertrophy* of the left ventricle, probably due to the fact that during diastole the blood is forced into the left ventricle by the over-distended auricle in large amount and under great pressure, so that the ventricle has more work to do.

The other results are the same as those of mitral stenosis.

Physical Signs.—1. On *inspection*, *palpation* and *percussion*, the evidences of enlargement of the heart.

2. On *auscultation*. A *systolic* sound heard most clearly at the *apex*, as a rule, but also *towards the left*, because the apex strikes the ribs, through which the sound is transmitted, and also at the

lower angle of the left scapula, or thereabout, because the current of blood is directed towards that point.

Accentuation of second sound at the pulmonary valves because of increased tension in the pulmonary vessels.

Diagnosis.—1. From *aortic obstruction* by the seat of the murmur and its area of diffusion.

2. From *tricuspid regurgitation* in the same way.

TRICUSPID REGURGITATION.

(*Tricuspid insufficiency*).

Symptoms and Results.—The *symptoms* and *results* are those of impediment to the venous circulation or passive hyperæmia; the most important are, (1) dropsy; (2) gastric catarrh; (3) dyspnœa; (4) renal disturbances, albuminuria, &c.

Physical Signs.—1. On *inspection*, over-distension of the veins with cyanosis venous pulsation, and usually feeble heart's action; dropsy, &c.

2. On *palpation*, venous pulse, (in jugular) hepatic pulsation and enlargement, weak heart's action.

3. On *percussion*, cardiac hypertrophy, hepatic enlargement, often ascites.

4. On *auscultation*, *systolic* murmur heard most distinctly at ensiform cartilage because the right ventricle is nearer to the examiner's ear there than elsewhere; diffusion slight, because the right ventricle is weak.

Diagnosis.—From (1) *aortic obstruction* and (2) *mitral regurgitation* by the seat of the murmur and the very limited diffusion.

General Course and Results of Valvular Disease.—The *course* and *results* of valvular disease has been explained in connection with disease of the individual valves.

Hypertrophy is probably the first thing which occurs, and that is compensatory in character.

Dilatation occurs at a later period, and is due to failure of compensation.

Relative Frequency of Valvular Diseases.—Valvular diseases stand in the following order, with respect to frequency:

1. Mitral regurgitation.
2. Aortic stenosis.
3. Aortic regurgitation.
4. Mitral stenosis.
5. Tricuspid regurgitation.

The others are extremely rare. As a matter of fact, *tricuspid regurgitation* occurs in the later stages of *all* the cardiac valvular

diseases which terminate fatally, so that while *primary* tricuspid regurgitation is rare, secondary trouble of this kind is very common.

It is very common, too, to have two or more valves involved simultaneously, such as mitral regurgitation and aortic stenosis, or aortic stenosis and aortic regurgitation, &c. On this account, it is not easy, in the late stage of valvular disease, to say which valve was primarily affected.

Prognosis of Valvular Diseases.—The *prognosis* of valvular diseases is dependent on, (1) the form of disease present; (2) the general health of the patient; (3) the degree of compensation.

Mitral obstructive disease usually runs a more rapidly fatal course than any other, because it rapidly causes over-filling of the pulmonary vessels, dilatation of the right ventricle and a general passive hyperæmia with its results—dropsy, &c.

Mitral regurgitant lesions stand next, perhaps, in point of gravity, but persons may live for years with this trouble, because the compensation is often well marked.

Aortic stenosis causes very distressing cerebral symptoms, but there is often well-developed compensation in these cases.

Aortic insufficiency gives the best prognosis because the compensatory hypertrophy can remedy the defect for years.

The prognosis of *tricuspid regurgitation* is very bad, but it should be remembered that this disease is very rarely primary, but usually results from some other serious valvular or pulmonary lesion.

A *complete cure* of valvular disease is apparently possible (Flint) and in most cases by judicious treatment symptoms of the most alarming character can be temporarily relieved.

Treatment.—A. *Prophylactic.* The administration of alkalis in acute rheumatism unquestionably prevents cardiac complications.

Over training for athletic sports and very violent physical exertion of all kinds is to be avoided.

B. When *compensation is good* the avoidance of over-exertion and excitement and abstinence with respect to stimulants are to be enjoined. Over-eating is also injurious. Strength should be maintained by nourishing food and tonics, such as iron, quinine, and especially strychnine.

6. When *compensation has failed* the indications are—

1. To *lessen the work of the heart* by the avoidance of exertion or excitement; prudence with respect to diet and stimulants, and the use of *nitro-glycerine*, which dilates the vessels and thus lessens the work of the heart.

2. To *increase the force of the heart* by (1) *digitalis*, which lengthens the diastole and thus *rests* the heart; if dropsy is not great *nitro-glycerine* and *digitalis* may be given together, the object of the *nitro-glycerine* being to lessen the work of the heart; (2) *strophanthus*; (3) *convallaria*; (4) *sparteine* and (5) *barium chloride* have

a very similar effect to digitalis. (6) *Strychnine* is a very useful heart tonic and is especially useful in aortic stenosis, when digitalis should be avoided if possible, because the prolonged diastole allows too much blood to enter the left ventricle. (7) Iron is a useful heart tonic, but is slow in its action.

3. To relieve symptoms such as dropsy, dyspnoea and insomnia.

Dropsy may be relieved by (1) *increasing the action of the kidneys* by digitalis, squills, calomel, &c.; (2) *the use of hydragogue cathartics*, such as calomel and jalap, elaterium and salines; (3) *the direct withdrawal* of water by aspiration or small punctures in the skin.

Dyspnoea is to be relieved by diminishing the dropsy, forcing the blood on through the lungs, and by the use of quebracho, codeia or morphia.

Insomnia is best treated by paraldehyde and codeia or morphia.

Oertel's treatment is intended to increase the power of the heart and to diminish dropsy. It consists essentially in *forced exercise* (mountain climbing) to the extreme limit of endurance and the diminution of the amount of fluid taken into the body. The method is of *very doubtful propriety* in any case, except one of fatty infiltration of the heart, and in all cases the utmost caution is required to prevent sudden overtaxing of the heart.

HYPERTROPHY OF THE HEART.

• (Enlargement of the heart.)

Causes.—The *essential cause* is over-work of the heart, which may be due to—

1. *Dilatation of the cavities*, which thus receive more blood, and consequently gives the muscular walls more work to do

2. *Mechanical obstruction* to the outflow of blood; this obstruction may be (1) at the valves, or (2) in the arteries.

Obstruction in the *systemic arteries* causes *left sided hypertrophy*, and is usually due to *arterio-capillary fibrosis* and atheroma, and is common in Bright's disease; it may also be due to pressure on a large vessel from without by a tumor, for example, or to a congenital narrowing of the aorta (rare).

Obstruction in the *pulmonary arteries* leads to hypertrophy of the *right side* of the heart, and is usually due to emphysema, in which many of the blood vessels in the lungs are destroyed, and the right ventricle has to do more work to force the blood through those which remain.

3. *Excessive functional activity* of the heart, as in prolonged and frequent attacks of palpitation, may ultimately lead to hypertrophy.

Morbid Anatomy.—1. The *size* of the heart is increased.

2. The *shape* depends upon the seat of hypertrophy; if the left ventricle is enlarged, the heart is triangular; if the right is chiefly affected it is more globular, because the thin walls of the right ventricle allow it to distend in all directions.

3. The *position* depends upon the seat of enlargement also; in *left-sided* hypertrophy the apex beat is moved very far to the left of its normal position; in *right-sided* hypertrophy the dullness on the right of the sternum is increased in extent.

4. The *thickness of the walls* is increased to a greater or less extent (see Valvular Lesions).

Symptoms.—The *symptoms* are due chiefly to the disease causing the hypertrophy; the *pulse*, except in aortic stenosis, is full and strong and hard, the face red, and the intra-vascular tension is increased.

Apoplexy is liable to occur in these cases if atheroma is present. Nose bleed is common and often excessive.

Cough occurs in a number of cases, and is probably due to the congestion of the lungs, which occurs in cases of mitral disease.

Physical Signs.—The *area of cardiac dullness* is increased. In health, the cardiac dullness is triangular in shape, one angle of the triangle being at the mid-sternal line, opposite the upper borders of the fourth costal cartilages, another angle at the apex, and the third in the middle of the sternum, on a line with the apex.

The *apex beat* is from $3\frac{1}{2}$ to $3\frac{3}{4}$ inches to the left of the seventh costo-sternal articulation.

In hypertrophy, the dullness on percussion frequently extends from the left nipple, or even beyond it, to an inch to the right of the sternum.

The *heart sounds* on auscultation are heard with unusual distinctness.

Diagnosis.—It is to be distinguished from *pericarditis* with effusion (q. v.).

Prognosis.—The *prognosis* depends upon the cause, and the general vigor of the patient.

Treatment.—The *treatment* is practically that of valvular disease, when compensation is good.

Aconite is of service if the force of the heart's beat is very great.

DILATATION OF THE HEART.

Definition.—Stretching and enlargement of one or more of the

cavities of the heart. It is nearly always preceded by hypertrophy, and is due to *failure of compensation*.

Causes.—1. Anything which *weakens the tone* of the cardiac muscle, such as peri- or endo-carditis, fatty degeneration or prolonged disease, such as typhoid fever (Loomis).

2. *Increased internal pressure* during diastole, which may be due to (1) obstructive, or (2) regurgitant valvular disease.

Morbid Anatomy.—1. The most common *seat* is the right ventricle, but the left auricle is often dilated, and all the cavities may be.

2. The *muscular fibres* are usually degenerated, fatty or albuminoid.

Symptoms.—The *symptoms* are those of failing compensation, (see Causes and Results of Valvular Diseases).

Physical Signs.—On *percussion*, the area of cardiac dullness is found to be increased.

On *auscultation*, the sounds are feeble.

Prognosis.—The prognosis is bad, but temporary improvement may be obtained from the use of digitalis, &c.

Treatment.—That of valvular disease, when compensation is failing.

MYOCARDITIS.

(Inflammation of the muscular structure of the heart.)

Myocarditis is of comparatively rare occurrence.

Causes.—1. *Disease of the coronary arteries* (atheroma and arterio-sclerosis, q. v.), which leads to degeneration and atrophy of muscular tissue and secondary growth of connective tissue, as in secondary degeneration of the spinal marrow.

Endocarditis and *pericarditis*, in which there is some infiltration of the adjacent muscular tissue leading to formation of connective tissue from the leucocytes.

Morbid Anatomy.—1. The chief *seat* of myocarditis is the left ventricle; it occurs in patches, probably because some branches of the coronary artery only, are atheromatous.

2. The *structural changes* consist in the disappearance in patches of the muscular tissue and its replacement by connective tissue, which has a white appearance.

Complications and Results.—*Endo-carditis* is a frequent complication from the extension of the inflammatory process inward to the endothelial surface.

2. *Cardiac thrombosis* may result from the death of the endothelial cells at the inflamed spot.

3. *Cerebral embolism* may occur from the lodgment of a piece of the thrombus in a cerebral vessel.

4. *Cardiac aneurism* is an occasional sequel, because the loss of muscular tissue weakens the wall of the heart at some point, and it dilates in consequence.

5. *Rupture of the heart* is a very rare result of myocarditis.

Symptoms.—The *symptoms* are similar in all respects to those of valvular disease with failing compensation. Angina pectoris often occurs.

Physical Signs.—The *physical signs* are similar to those of dilatation; valvular murmurs are *absent*.

Diagnosis.—It is distinguished from (1) valvular disease by the absence of murmurs; (2) fatty degeneration (with great difficulty) by the existence of atheroma.

Prognosis.—The *prognosis* is uniformly unfavorable.

Treatment.—The *treatment* is that of failing compensation.—

1. To lessen the work of the heart.

2. To *tone up the heart* as far as possible. Strychnine and arsenic are the most useful remedies for this purpose; cod liver oil is also of service. Digitalis should be given with great caution, because some of the muscular tissue has been lost, and that which remains may readily be over-stimulated.

3. To *relieve symptoms*, such as dyspnoea, dropsy and pain. (See treatment of valvular disease and angina pectoris).

FATTY DEGENERATION OF THE HEART.

Varieties.—1. *Fatty infiltration*, in which the fat lies between the bundles of muscular fibres.

2. *Fatty metamorphosis*, in which the protoplasm of the muscle cells is *replaced* by fat.

Causes.—The *chief cause* of *fatty infiltration* is (1) the excessive use of starchy or saccharine food, and malt liquors, and (2) sedentary habits.

The *chief causes* of fatty degeneration proper (metamorphosis) are (1) wasting diseases, such as phthisis and typhoid fever, (2) old age in which metabolism is defective and there is often disease of the coronary arteries, (3) anæmia from any cause; the essential cause probably is defective oxidation. The causes are often unknown.

Morbid Anatomy.—In fatty *infiltration* there is simply an accumulation of fat upon the heart and between the bundles of muscular fibres.

In fatty *metamorphosis*, the *color* of the heart is paler than normal, and the *consistence* is less. The fat appears as small globules scattered through the muscular fibres.

Symptoms.—1. *Circulatory.* The pulse is weak and irregular, the extremities are cold, and attacks of syncope may occur; these symptoms are due to weakness of the pump (the heart) and consequent defective circulation.

2. *Respiratory.* Breathlessness on exertion is very marked because of the weakness of the heart and the insufficient supply of blood to the lungs in consequence. *Cheyne-Stokes breathing* often occurs; its cause is unknown.

3. *Cerebral.* The cerebral symptoms are, vertigo and want of decision from insufficient supply of blood to the brain.

Physical Signs.—The *physical signs* are not usually marked. A weak impulse on palpation, and feeble sound on auscultation, with some increase of the extent of cardiac dullness (due to dilatation) are all.

Diagnosis.—A certain *diagnosis* from *simple* dilatation can only be made by the history of the case, ordinary dilatation being preceded by hypertrophy. A diagnosis from myocarditis is practically impossible.

Prognosis.—In fatty *infiltration* the prognosis is not hopeless if a proper mode of life be adopted. In fatty *metamorphosis* recovery is out of the question, as a rule, except when it occurs in acute diseases.

Treatment.—The indications and the means of fulfilling them are in the main the same as in myocarditis.

NEUROSES OF THE HEART.

(Functional disorders of the heart.)

Varieties.—The most common varieties of functional diseases of the heart are—

1. *Palpitation*, in which the heart frequently acts with great rapidity, and often irregularly for a few moments at a time.

2. *Intermittence*, in which a beat is lost occasionally. This is very common, and of little or no significance.

3. *Tachycardia*. This affection is rare; it is characterized by attacks of extremely rapid action of the heart, the pulse being often from 160 to 200 per minute.

4. *Irritable Heart*. This affection is also a rare one; it is characterized by palpitation and præcordial pain, and usually occurs after exertion, but may be independent of it.

Causes.—The common causes of functional affections of the heart are—(1) the presence in the blood of certain poisons, such as nicotine; (2) excessive irritability of the cardiac nerves or the muscular tissue itself; (3) disturbances of the digestive organs, which set up a reflex disturbance through the pneumogastric nerves; (4) anæmia, which impairs the power of the heart and increases the liability to palpitation, &c.

Diagnosis.—Functional heart diseases are distinguished from organic by the absence of any of the signs of the latter on physical examination.

Prognosis.—The *prognosis* is always favorable, but if increased functional activity is of long duration it *may* lead to hypertrophy (q. v.).

Treatment.—The first indication is to *remove the causative condition*, such as dyspepsia, flatulence, anæmia, &c.

2. To *quiet the heart's action*, *cardiac sedatives*, such as Hoffmann's anodyne, valerian, camphor, musk, aromatic spirits of ammonia and the bromides, are indicated.

ANGINA PECTORIS.

(Neuralgia of the heart—Stenocardia.)

Causes.—1. Anything which *increases the intra-vascular pressure*, such as (1) spasm of the vessels; (2) atheroma.

2. Any *obstruction to the discharge of blood* from the heart, such as aortic stenosis.

3. Anything which *impairs the muscular power* of the heart, so as to cause a *loss of balance* between the muscular power of the heart and the vascular tension, such as myocarditis, fatty degeneration, loss of compensation in valvular disease.

4. It occurs usually in advanced life (see Atheroma.)

Morbid Anatomy.—There is none which is characteristic (see Causes.)

Symptoms.—1. *Nervous.* Pain extremely severe in character, situated in the præcordial region and often running down the *left* arm, rarely the *right* is the most prominent symptom. This pain occurs in *paroxysms*, which rarely last longer than an hour, and may be many months or only a few hours apart.

2. *Respiratory.* There is great respiratory distress, but no actual difficulty in either inspiration or expiration.

3. The *skin* is usually pale from the arterial spasm or weakness of the heart.

4. *Circulatory.* The *pulse* may be hard and rolling (in atheroma) or feeble when there is fatty degeneration without marked atheroma of the vessels.

Diagnosis.—It is distinguished from—1. *Spasmodic asthma*, by the fact that there is no real difficulty in expiration, and pain in asthma is not marked.

2. *Intercostal neuralgia* by the absence of tender points in angina pectoris.

3. *Simple hysteria* by the general history of the case. (The violent præcordial pain which sometimes occurs in hysteria is probably due to spasm of the blood vessels).

Prognosis.—The *prognosis* in genuine angina pectoris occurring in connection with atheroma, or degenerative changes in the cardiac muscle, is always unfavorable. In cases connected with hysteria the prognosis is favorable.

Treatment.—*During the paroxysm* the indications are—

1. To *relax spasm* of the vessels.
2. To *relieve pain*.
3. To *increase the power of the heart*.

Amyl nitrite given by inhalation relaxes spasm far more promptly than any other drug; it fails to give relief in those cases when the arteries are atheromatous over an extensive area, and when the chief difficulty is loss of heart power.

Morphia, hypodermically, is the best agent to relieve pain.

Brandy, coffee, digitalis, ether and camphor may be used to increase the power of the heart.

In the interval between the paroxysms, the indications are—

1. To *prevent spasm* of the vessels.
2. To *increase the power* of the heart.

Nitro-glycerine (or some other nitrite) is by far the best drug to prevent spasm of the vessels; it fails in those cases which are due to extensive atheroma.

Digitalis is sometimes useful, but must be given with caution in degeneration of the heart muscle.

Strychnine and arsenic increase the power of the heart; iron has a similar action. Iodide of potassium is advised by Huchard.

ATHEROMA.

(Chronic Endarteritis.)

Causes—1. *Age and sex*—The disease is most common in elderly men because they are most exposed to the other causes such as—

2. *Overstrain* of the heart from prolonged work of a character to increase the force of the heart's action.

3. *Alcoholism, rheumatism and gout*; these diseases probably cause atheroma because the morbid matter circulating in the blood in them acts as a direct irritant to the vascular wall.

Morbid Anatomy.—1. The most common *seat* is in the aorta because it is subjected to more strain than other vessels. Another common seat is in the *cerebral* vessels; it also occurs in the femorals, radial, ulnar, &c.

2. *Structural changes*.—The disease occurs in patches of greater or less size and the interna is chiefly involved, the inflammation leading to the formation of calcareous plates.

Symptoms and Results.—The hardened arteries roll under the finger.

1. Hypertrophy of the left venticle results from increased work.

2. *Defective arterial circulation* and sometimes dry gangrene from narrowing of the vessel.

3. *Cerebral apoplexy* or thrombosis from bursting of a vessel or formation of a thrombus in a calcareous patch.

ANEURISM OF THE THORACIC AORTA.

Causes.—*Atheroma* is by far the most common cause of aneurism in this locality.

Morbid Anatomy. 1. The usual *seat* is in the ascending part of the arch, probably, because this part is subjected to great strain at each ventricular systole.

2. *Changes* in the walls of the vessel. The vascular wall

becomes thinner at the site of the aneurism from stretching; but it may be thickened secondarily by inflammatory action around it.

Symptoms. The *symptoms* briefly stated are of two kinds.

1. There is a *pulsating tumor* corresponding with some part of the aorta; often, generally in fact, a *murmur* can be heard over this tumor and sometimes a *thrill* can be felt. Sometimes it beats just as the heart.

2. *Pressure symptoms*, such as *dyspnoea* from pressure on the trachea, *pain* from pressure on the nerves, *passive hyperæmia* from pressure on the vena cava and other symptoms produced in the same way are always present and the combination of pressure symptoms and a pulsating tumor shows the nature of the disease (Da Costa).

Diagnosis. It is distinguished from *cancers and abscess* by the absence of marked expansive pulsation in the latter; from valvular disease of the heart by the *situation* of the murmur, if one is present and by the *seat* of the enlargement.

Prognosis.—The *prognosis* is always bad and recoveries are extremely rare.

Results.—1. *Coagulation of blood* in the sack is the first result when an aneurism is formed. The extent of this coagulation varies in different cases.

2. *Erosion* of the *ribs*, or of the vertebral column, often occurs at a later stage.

3. *Rupture* of the *aneurism* and death from hemorrhage is usually the final result; such rupture may occur into (1) the pericardium; (2) a bronchus; (3) the œsophagus; (4) the pleural cavity; (5) the vena cava.

4. *Recovery* is possible from coagulation of blood in the sack and gradual closure of the cavity in this way.

Treatment.—The *treatment* which has given the best results consists in absolute quiet in the recumbent posture; a very small allowance of food, and the administration of *iodide of potassium*.

Surgical treatment has not given good results, nor has the electrical treatment—the insertion of a needle connected with a galvanic battery into the sack to produce coagulation.

CHAPTER VI.

DISEASES OF THE KIDNEYS.

ALBUMINURIA.

(Albumin in the urine.)

Divisions and Causes of Albuminuria.—1. *False*, in which the albuminuria is due to (1) *suppuration* somewhere in the urinary tract, as occurs in cystitis and gonorrhœa, or (2) hemorrhage, in which the albumin is poured into the urine along with the other constituents of the blood.

2. *True* albuminuria, which may probably be caused by one of the following conditions—

(1) *Alterations in blood pressure* which probably explains those cases of albuminuria occurring after violent exercise or at certain hours of the day (cyclical albuminuria);

(2) *Changes in the vascular walls*, as in waxy degeneration of the kidneys;

(3) *Changes in the composition of the blood* which may act in two ways—first by making the blood itself more apt to pass through a membrane, and secondly, by rendering the membrane more permeable to the albumin by defective nutrition of the epithelial cells;

(4) *Degeneration of renal epithelium*, which is by far the most important cause, and occurs in inflammatory affections and probably also in connection with waxy degeneration.

Tests for Albumen.—1. *Heat* causes coagulation to a greater or less extent provided the urine is acid.

2. *Nitric acid* added to the urine precipitates the albumen.

Heat causes precipitation of earthy phosphates also, but these are at once dissolved on the addition of an acid; if there is a precipitate with *both* nitric acid and heat, albumen is certainly present in the urine.

Significance of Albuminuria.—The *occasional* occurrence of albumen in small quantity in the urine after violent exercise or a meal rich in albuminous food is entirely consistent with health. If it is *present persistently* and in considerable quantity it is always a serious symptom, because it shows that the epithelial cells are incapable of doing their work (renal inadequacy) and that *urinary solids are retained in the blood*.

It should be distinctly understood, however, that the *absence* of albumin from the urine is *not* an evidence that the kidneys are sound.

Quantitative Test for Urinary Solids.—The amount of urinary solids passed in twenty-four hours may be determined with reasonable accuracy in a very simple way.

Multiply the last two figures of the specific gravity of the urine by the number of ounces passed in twenty-four hours; the result will be the amount of urinary solids expressed in grains.

Normal sp. gr. = 1020; normal amount of urine passed in twenty-four hours about 48 oz.; amount of solids, 960 grains.

TUBE CASTS.

Varieties.—1. *Hyaline*, which are composed of albuminoid substance which has coagulated in the urinary tubules and appear in the urine as glassy or homogeneous cylinders from $\frac{1}{2500}$ to $\frac{1}{500}$ of an inch in diameter. In some cases probably the casts consist of an inflammatory exudate, but the nature of the material in many cases is unknown.

2. *Epithelial*, which are merely hyaline casts on the surface or in the substance of which are epithelial cells from the urinary tubules.

3. *Blood casts* are sometimes merely *coagula* of blood formed in urinary tubules in cases of hemorrhage, and at other times a *hemorrhagic exudate* occurs in inflammation of the kidneys and leads to the formation of blood casts.

4. *Fatty casts* contain epithelial cells which are undergoing fatty degeneration, or fatty particles formed by the breaking up of such cells.

5. *Granular* are formed by the breaking up of epithelial cells without fatty degeneration.

Significance of Casts.—Several kinds of casts are usually found in the same specimen of urine.

Hyaline casts, especially when of small size, are usually least serious in significance.

Epithelial and blood casts usually appear in recent cases of considerable severity.

Fatty and granular casts usually appear in chronic cases.

ACUTE URAEMIA.

Uræmic poisoning.

Varieties.—1. Eclampsia or convulsive form.

2. Comatose form.

The comatose is the most common except in puerperal cases.

Causes.—The *essential cause* is the retention in the blood of morbid matters which should be removed by the kidneys (loss of efficiency of renal epithelium).

It may occur in the various forms of nephritis (or Bright's disease q. v.), in waxy degeneration of the kidneys, often classed with Bright's disease, in cancer and tubercle.

Symptoms.—A. *Premonitory.* 1. *Nervous.* Headache, dullness, disturbances of vision, from retention of morbid matters in the blood.

2. *Digestive.* Nausea and vomiting, and sometimes diarrhoea, from the action of urinary solids on the nerve centres and also on the intestinal mucous membrane.

3. *Respiratory.* Attacks of dyspnoea (renal asthma) probably from the action of the impure blood on the respiratory centres.

4. *Urinary.* The *amount* of urine is usually greatly diminished, probably because the exudate around the glomeruli compresses the vessels and interferes with the discharge of water or the solids in solution therein.

The sp. gr. is usually high, about 1030, because the amount of water is both actually and relatively increased.

The *total amount* of solid urine is always greatly diminished.

Albuminuria and tube casts are always present.

B. *During the attack.* 1. In the *convulsive* form there is loss of consciousness, clonic convulsions, interrupted breathing, irregularity of the pulse, lividity of the skin and foaming at the mouth. A convulsive attack usually lasts a few moments only, but they usually recur at intervals of a few hours, and there is unconsciousness during the intervals.

2. In the *comatose* form there is profound unconsciousness, stertorous breathing, a full and slow pulse and usually contraction of the pupils.

Diagnosis.—It is distinguished from, 1, *Epilepsy*, by the history of the case and the urinary symptoms. 2, *Apoplexy*. 3, *Hysteria*. And 4, *Opium poisoning* by the presence of albumin and casts in the urine.

Prognosis.—The *prognosis* is always serious, but in cases of acute Bright's, especially the puerperal form, recovery generally occurs.

Treatment.—A. *Prophylactic.* 1. The *diet* should be poor in nitrogenous food except *milk*, which may be given freely; it washes out the kidneys.

2. *Diuretics* are not advisable, except *two*, *water* or lithia water and *digitalis*; these directly increase the flow of water from the kidneys but do not stimulate the cells.

3. To *remove waste matters* by the *skin* and *bowels*.

To *induce sweating*, the hot pack, steaming and pilocarpine may be employed.

To *cause watery actions* from the bowels, calomel, jalap, elaterium and the saline cathartics are used.

B. *During the paroxysm*, if of *convulsive* form—

Chloroform by inhalation, chloral by enema, the bromides in the same way, and morphia hypodermically, give the best results. Antipyrine, amyl nitrite and other agents to allay nervous irritability or to relax spasm, have been employed.

CIRCULATORY CHANGES WHICH ARE COMMON IN RENAL DISEASES.

Changes in the Blood Vessels.—In all or nearly all cases of Bright's disease there is more or less *thickening of the walls* of the smaller blood vessels due probably to inflammation set up by the circulation in the blood of some irritating substance or substances. This change is frequently called *fibroid degeneration* of the vessels or *arterio-capillary fibrosis*. The "fibroid" or connective tissue is formed from the leucocytes which pass out of the vessels in inflammation.

Changes in the Heart.—*Hypertrophy* of the heart occurs from the increased work put upon the heart in consequence of the changes in the blood vessels and the high arterial tension caused thereby.

Dilatation occurs later on, just as in other cases of cardiac hypertrophy.

Character of the Pulse.—As a rule, the *pulse*, in diseases of the kidneys, is slow, full and strong—the pulse of increased arterial tension; the cause of this is evident from the changes in the heart and vessels. When *dilatation* of the heart occurs, however, from failure of compensation, the pulse becomes quick and feeble.

Influence of Circulatory Changes on the *amount* of urine discharged.—As a rule, the greater the blood pressure, the greater the amount of urine discharged, unless there is counter-pressure on the

vessels of the glomeruli by an inflammatory exudate. It follows, therefore, and is in accordance with clinical experience, that in *parenchymatous nephritis* when there is much exudate the *amount of urine is diminished in spite of the increased arterial tension*; while in *interstitial nephritis*, when the connective tissue framework is chiefly involved, the *amount of urine is increased*.

This difference in the pathological condition in parenchymatous and interstitial nephritis explains also the fact that *dropsy* is a common symptom in parenchymatous disease and is rare or slight in the interstitial form. *Waxy degeneration* comes between parenchymatous nephritis and interstitial nephritis with respect to the amount of urine discharged and the degree of dropsy; if the glomerular vessels are chiefly involved it approaches parenchymatous nephritis in symptoms, while if other vessels are chiefly involved the dropsy is less marked and the urine more abundant. It must be remembered also that in waxy degeneration there is generally great muscular weakness from the accompanying conditions and the vascular tension may be diminished in consequence.

RENAL HYPERÆMIA.

Varieties.—1. Active.

2. Passive.

Active hyperæmia is usually the first stage of an inflammatory process and is practically indistinguishable from nephritis, except by its short duration.

The probable *causes* are exposure to cold and the use of renal irritants, such as turpentine and cantharides.

The *symptoms* are pain in the back, headache and scanty and dark-colored urine, which contains albumin and often blood or hyaline casts.

The *diagnosis* is based on the rapid improvement under appropriate treatment.

The *prognosis* is usually favorable, but the fact should be borne in mind that this affection is often the commencement of nephritis.

The *treatment* consists in dilating the vessels of the skin by warm baths, pilocarpine, amyl nitrite or nitro-glycerine, and in washing out the kidneys by administering large quantities of pure water or of lithia water.

PASSIVE HYPERÆMIA.

Causes.—Venous stasis (passive hyperæmia) from heart disease, certain pulmonary affections, as emphysema, and pressure on the veins by the gravid uterus, dropsical effusion or tumors.

Morbid Anatomy.—1. The *size* of the kidneys is increased from the amount of blood and the exudate or transudate which occurs.

2. The *color* is dark and the *consistence* firmer than normal, in old cases especially, because—

3. There is an *increase* of *connective tissue* which is formed from the leucocytes which have traversed the vessels (see Results of passive hyperæmia).

4. *Cloudy swelling*, or albuminoid degeneration of the renal epithelium, also occurs from a deficient supply of arterial blood.

Symptoms.—The *pulmonary*, *digestive* and *nervous* symptoms are those of venous stasis from any cause.

The *urinary* symptoms are *scantiness* from diminished arterial tension, *dark color* from the presence of blood or of blood-coloring matter and high specific gravity because the water is diminished more than the salts.

Albumen and *casts* are usually present.

Diagnosis.—The *diagnosis* is based on the causative condition.

Prognosis.—The *prognosis* is usually unfavorable because it occurs usually as a result of incurable heart or lung trouble.

Treatment.—The chief aim is to *relieve the passive hyperæmia* and *increase the arterial tension*. Digitalis is by far the best remedy for this purpose.

BRIGHT'S DISEASE.

A number of different affections of the kidneys resembling each other, however, in certain particulars, have been included under the general name of Bright's Disease.

Classification.—The following *classification* is sufficient for practical purposes—

1. *Parenchymatous inflammation* of the kidneys in which the cells of the renal tubules are *chiefly* involved. It may be either (1) *acute* or (2) *chronic*.

2. *Interstitial inflammation* of the kidneys, in which the part chiefly affected is the connective tissue framework of the organs. It is *always chronic* in course.

3. *Lardaceous* or *amyloid* or *waxy degeneration* of the kidneys; in this form the walls of the smaller blood vessels are primarily and chiefly involved; the affection is *degenerative* and *not inflammatory* in character, and runs a *chronic* course.

ACUTE BRIGHT'S DISEASE.

(Acute nephritis; acute desquamative nephritis; acute parenchymatous nephritis.)

Definition and Frequency.—An acute inflammation of the kidneys, in which the parenchyma is chiefly involved, and which is of common occurrence.

Causes.—1. *Sudden chilling* of the body; the mode of action of this cause is unknown; it is probably connected with the increased work thrown upon the kidneys when the vessels of the skin are contracted.

2. The *acute* (rarely the chronic) *infectious diseases*; the inflammation is probably due to the action of the leucomaines on the kidneys, by which they are in part eliminated. The disease is especially common after scarlet fever, but may also occur as a sequel of diphtheria, typhoid fever, cerebro-spinal meningitis and the other acute infectious diseases.

3. Certain *irritants*, such as turpentine and cantharides, which are eliminated by the kidneys.

4. *Pregnancy* is an occasional cause; excessive work and the elimination of irritants is the probable explanation in these cases.

Morbid Anatomy.—1. The *size* of the kidneys is more or less increased, from infiltration with the exudate.

2. The *color* is *grayish* because the pressure of the exudate forces the blood out of the vessels; but often red spots are seen from the congestion of the glomeruli or a bloody exudate into Bowman's capsule.

3. This *capsule* is often filled with an exudate which compresses the vessels in the glomeruli.

4. The *epithelium* of the convoluted tubules is cloudy or may be peeling off, and the *tube itself* is often filled with the exudate and desquamated cells.

Symptoms.—1. *Cutaneous.* Dropsy is usually the most prominent symptom; it is most marked about the face (See Dropsy in General Pathology) and is in large amount because the plugging of Bowman's capsule and the convoluted tubules interferes with the discharge of water by the kidneys. The *skin* is usually pale from the action of the retained water on the red corpuscles.

2. *Urinary.* The *amount* of urine is diminished from the pressure of the exudate on the vessels of the glomeruli; the *sp. gr.* is high—often 1030, because there is relatively a greater diminution of water than of solids; it contains *albumen* and *casts*, usually epithelial, hyaline and bloody, and the amount of urinary solids is diminished.

3. The *nervous* symptoms are those premonitory of uræmia (q. v.).

4. The *ocular* symptoms are rarely marked in acute Bright's, but there may be more or less *disturbance of vision* from the action of retained matters on the nervous system ; retinitis is rare in acute cases.

5. The *digestive* and *respiratory* symptoms are like those of threatened uræmia, and are due to the action of morbid matters on the nerve centres and the intestines, and also to the dropsical accumulation which interferes with breathing.

6. The *temperature* is rarely elevated above 102° and often is not over 100° .

Diagnosis.—The *diagnosis* is based on the headache, nausea and respiratory disturbances, but above all on the urinary symptoms and signs. *Scanty discharge, albuminuria* and the presence of *casts*.

Prognosis.—The *prognosis* depends on the *age* of the patient, the *cause* of the nephritis and the complications ; it is better in children, and especially after scarlet fever, than under other circumstances. Cases occurring in connection with pregnancy also have, as a rule, a favorable prognosis.

Complications.—The most common *complications* are pneumonia, pleurisy, pericarditis and endocarditis.

Causes of Death.—1. *Uræmia* (or urinæmic poisoning) from the retention of morbid matters in the blood.

2. *Pneumonia*. It is not clear how this disease results from acute Bright's.

3. *Dropsy*, such as hydrothorax and pulmonary œdema.

Treatment.—1. *Dietetic*. To *diminish* the work of the kidneys by giving nitrogenous food, *except milk*, in small quantities, and by increasing the action of the skin and bowels.

2. To *remove* the *urinæmic solids* and *relieve dropsy* by

(1) *such diuretics* as digitalis and water or lithia water ;

(2) *hydragogue cathartics* ;

(3) *diaphoretics*.

3. To *draw blood to the skin* and so lessen the amount in the kidneys by poultices, baths and warm clothing.

4. To *relieve symptoms* as they arise.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Bright's Disease.)

Definition and Frequency.—Chronic inflammation of the kidneys in which the parenchyma is chiefly involved. It is comparatively common.

Causes.—1. *Age and sex.* Men in middle life are most liable to this affection.

2. *Exposure* to cold and dampness seems to be a cause, and acts probably by increasing the work of the kidneys, but the exact connection between cold and nephritis is not plain.

3. *Alcohol* is undoubtedly a cause; it probably acts as a direct irritant to the renal cells.

In many cases no cause can be discovered.

Morbid Anatomy.—*Two forms* are described—

1. The *large white* kidney.

2. The *fatty and contracted* kidney.

They are different stages of the same disease.

1. The *size* is increased because of the exudate into the organ.

2. The *color* is *whiter* than normal because the blood is pressed out of the vessels by the exudate.

3. The *consistence* is firmer because the urinary tubules are more or less filled with a solid exudate.

4. The *capsule* is more or less adherent; the longer the disease has lasted the greater the adhesion.

5. On *microscopic examination* the capsules of Bowman are found more or less filled with exudate; and the vessels and glomeruli are surrounded in many places by connective tissue.

The *renal cells* lining the tubules are either *albuminoid* and *desquamating* at a comparatively early stage, or are *fatty* later on.

The *fatty and contracted* form is merely a later stage of the *large white* form, and the later the stage the more marked is the fatty degeneration and the greater the amount of connective tissue.

The kidneys are not uniformly affected, but the degenerative change is worse in some places than in others.

Symptoms.—1. *Cutaneous.* *Dropsy* is present to a greater or less degree; it is due to the counter-pressure of the exudate on the glomeruli which prevents the flow of water from the vessels. In very chronic cases the dropsy is slight because the exudate is small in amount and the blood pressure is greatly increased by the cardiac hypertrophy.

Pallor is usually a marked symptom, because of the hydræmic condition of the blood.

2. *Urinary.* *Albuminuria* is always present; the amount depends upon the activity of the inflammation. The *quantity* of urine is diminished in the earlier stages, but when cardiac hypertrophy occurs and the exudate diminishes it is increased.

The amount of *urinary solids* is always *less* than normal because of the loss of renal epithelium.

Casts—granular and fatty, chiefly—are always present.

3. The *nervous* and *digestive* symptoms are not usually marked unless an acute attack supervenes.

4. *Circulatory.* The heart is hypertrophied and the pulse is full and strong (see *Circulatory Changes in Renal Disease*).

5. The *special sense* symptoms. *Disturbances of vision* are of frequent occurrence and are commonly due to an exudate into or an atrophy of the retina which is plainly visible as a white patch on ophthalmoscopic examination.

Diagnosis.—The *diagnosis* is based on the urinary symptoms and signs and the dropsy.

Prognosis and Duration.—The *prognosis* is unfavorable, but the disease may run on in a very chronic state for months or years.

Complications.—1. *Pulmonary.* Pneumonia and pleurisy are frequent complications. They are due to the irritation of waste products retained in the blood.

2. *Inflammation of serous membranes* generally; endocarditis, pericarditis and synovitis occasionally occur.

3. *Cardiac hypertrophy* is an invariable complication of cases of old standing. The way in which it is brought about has already been explained.

Treatment.—The indications are—

1. To *clear out the tubes* by the administration of digitalis and the use of large quantities of water.

2. To *lessen renal hyperemia and the work of the kidneys* by dilating the vessels of the skin by warm baths, jaborandi and nitroglycerine; to administer only small quantities of nitrogenous food except milk, which may be given freely

3. To *improve nutrition* by cod liver oil (Loomis), iron and strychnine.

4. To *relieve symptoms*, such as dropsy, by diaphoretics, hydragogue cathartics, digitalis, small punctures and the aspirator.

Headache and sleeplessness are best relieved by phenacetine and paraldehyde.

INTERSTITIAL NEPHRITIS.

(Cirrhotic Bright's Disease.)

Definition.—A very chronic affection of the kidneys in which the organ becomes progressively smaller and harder than normal from atrophy of its parenchyma and increase of connective tissue.

Causes.—Often no cause can be assigned; the disease occurs in advanced life, and chiefly in men. *Alcohol* and *lead*, *gout* and *rheumatism*, are supposed to be causes in some cases. All of these substances act as irritants to the renal cells during their elimination by that organ.

Morbid Anatomy.—1. The *size* of the kidneys is very much less than normal.

2. The *shape* is irregular and nodulated from the contraction of the new formed connective tissue.

3. The *consistence* is much firmer than natural for the same reason, and the *capsule* is closely adherent.

4. On *microscopical* examination many of the glomeruli and urinary tubules are found atrophied and completely destroyed; those which remain are usually sound. There is an enormous increase in the connective tissue in the organs.

5. *Changes in other organs.* Arterio-capillary fibrosis and hypertrophy of the heart are always present and in many cases there is cirrhosis of the liver.

Symptoms.—1. The onset is very gradual and insidious, and the symptoms are obscure.

2. *Urinary.* The *amount* of urine is increased; the specific gravity is low; these changes are due to the hypertrophy of the heart and the absence of an exudate from the renal tubules. *Albumin* may be present in small quantity, but is often absent, because the glomeruli which remain are usually sound—those which are affected at all are entirely destroyed. The amount of urinary solids is always diminished.

3. *Cutaneous.* *Dropsy* is never marked because there is no exudate in the tubules and the blood pressure is increased. Some dropsy in the ankles appears late from heart failure.

4. *Headache* and *vertigo* and sometimes local paralysis appear; the cause of these symptoms is not clear; probably they are connected with the retention of urinary solids.

5. *Dyspeptic* symptoms are also of common occurrence, and very distressing at times.

Diagnosis.—The *diagnosis* from *diabetes* is made by the absence of sugar from the urine.

A *constructive* diagnosis is often extremely difficult; it is based on (1) the increased flow of urine of low specific gravity; (2) the cardiac hypertrophy; (3) the nervous symptoms; and (4) the digestive disturbances.

Prognosis.—The *prognosis* is always *bad*; the disease is progressive; the duration often months or years. *Sudden uræmia* may occur from extension of disease to the remaining parenchymatous tissue, or death may result from complications.

Complications—1. *Cardiac hypertrophy* and arterio-capillary fibrosis have already been mentioned.

2. The *pulmonary* complications are similar to those of chronic parenchymatous nephritis with the addition of emphysema.

3. *Cerebral hemorrhage* occasionally occurs from disease of the vessels and hypertrophy of the heart.

Treatment.—The *treatment* is chiefly symptomatic; the previous disease (gout, rheumatism, &c.) should receive attention; alkaline waters should be administered; the blood pressure should be lowered by nitro-glycerine; and headache, insomnia and other symptoms should be relieved as far as possible by phenacetine, sulphonal, &c.

WAXY DEGENERATION OF THE KIDNEYS.

(Lardaceous or amyloid degeneration.)

Causes.—1. Syphilis. 2. Suppuration. (See Amyloid Degeneration in General Pathology).

Morbid Anatomy.—1. The *size* of the kidneys is greater than normal.

2. The *color* is pale; the *consistence* firm.

3. The *seat* of the degeneration is first on the walls of the blood vessels, but the renal cells and connective tissue may be involved later.

4. *Microscopic appearances.* The cells swell up and lose their outline; if tincture of iodine be applied they assume a mahogany color.

Symptoms.—The symptoms are very variable and not characteristic.

1. *Urinary.* The *amount* of urine may be increased, but it is often diminished because of the general debility and lessening of blood pressure in consequence. The *specific gravity* varies with the amount passed. *Albumin* is sometimes present in large quantity when the glomeruli are involved, but is absent if the vasa recta only are diseased. The amount of *urinary solids* is but little lessened and there are *few casts*.

2. *Dropsy* is not usually very marked, because there is no exudate into Bowman's capsules or the urinary tubules to prevent the discharge of fluid by the kidneys.

3. *Nervous* symptoms are not prominent because the amount of urinary solids is not much diminished.

Diagnosis.—The *diagnosis* is based on the urinary symptoms and the occurrence of amyloid disease in other organs.

Prognosis.—The prognosis is bad; the duration long.

Complications.—Waxy degeneration of the bowels, liver, etc.

Treatment.—That of the causative disease.

PYELITIS.

(Inflammation of the pelvis of the kidney.)

Causes.—1. *Stone* in the pelvis of the kidney.

2. *Irritating drugs*, such as carbolic acid, turpentine and cantharides, which are eliminated by the kidneys.

3. *Extension* from the bladder in cases of cystitis.

4. *Obstruction to the outflow of urine* leading to ammoniacal decomposition and consequent inflammation.

Morbid Anatomy.—1. *Redness and swelling* of the mucous membrane.

2. An *exudate* which is muco-purulent in character on the surface of the mucous membrane.

3. Sometimes *hemorrhagic extravasations* in the mucous membrane.

Symptoms and Signs.—1. *Nervous.* *Pain* in the region of the kidney and *frequent desire to urinate* are common symptoms, and are due to the hyper-irritability of the sensory nerves from the inflammation.

2. *Rigors and fever* may occur from the absorption of leucomaines.

3. The urine contains *pus* and "tailed" epithelial cells.

Diagnosis.—The *diagnosis* is based on the symptoms and the shape of the epithelium.

Prognosis.—The *prognosis* depends on the cause; it is good if the cause can be removed.

Treatment.—1. The first indication is to *remove the cause*.

2. To *dilute the urine* by giving the patient large quantities of water or of lithia water.

3. To *relieve the inflammation* by copaiba, cubebs, chlorate of potash, eucalyptus, &c.

HYDRONEPHROSIS.

(A collection of fluid in the pelvis of the kidney.)

Causes.—1. *Internal obstruction* by a calculus.

2. *External pressure* on the ureter by a tumor, such as cancer or possibly ovarian tumor.

Morbid Anatomy.—1. *Changes in the kidney.* The kidney becomes greatly distended and atrophies.

2. *The contents of the sack*, which may be as large as one's head, are at first urine simply, but later on the fluid contains a larger proportion of water.

Symptoms.—The essential symptom is a *tumor* in the region of the kidney.

Diagnosis.—It is *diagnosed* from an *ovarian cyst* by the different situation of the tumor.

Prognosis.—The *prognosis* is serious, but spontaneous recovery may ensue.

Treatment.—1. The *emptying of the sack* by (1) manipulation; (2) aspiration.

2. *Surgical measures*; removal of the sack if it is due to a stone.

HÆMATURIA.

(Blood in the urine.)

Causes.—Blood in the urine may come from—

1. The kidneys.
2. The bladder.
3. The urethra.

Rarely from the ureter.

Symptoms.—If the blood has come from *the kidney* it was probably poured out in consequence of passive congestion, acute inflammation, or the presence of a calculus, and the urine will be *smoky* in color.

If the blood has come from the *bladder* it is often in considerable amount and forms a clot which may interfere with the discharge of urine.

If the blood comes from the *urethra* it flows during the intervals between urinating.

Diagnosis.—The *diagnosis* of blood is made by the microscope; the diagnosis of the cause and seat by the accompanying conditions.

Prognosis.—The *prognosis* depends upon the cause; the hemorrhage in itself is not serious.

Treatment.—In cases of hæmaturia from the kidneys—ergot, gallic acid and tannic acid have been employed; in paroxysmal malarial hæmaturia, quinine is indicated.

Hemorrhage from the bladder and urethra belongs to surgery

CHYLURIA.

(Chyle in the urine; fatty urine.)

Causes.—This disease is confined for the most part to tropical countries and is due to the presence in the body—in the thoracic duct probably—of the *filaria sanguinis hominis*—a worm about three inches in length and very narrow.

Symptoms.—The *urine* is white and milky in appearance and is rendered clear on the addition of ether, which dissolves the fat.

The *perspiration* is sometimes cyhlous also.

Diagnosis.—The *diagnosis* is made by examination with the microscope and by adding ether to the urine, which renders it clear.

Prognosis.—The *prognosis* is serious, and death may be brought about by anæmia; but the duration is usually long.

Complications.—*Elephantiasis* is a common complication and seems to be due to the same cause.

Treatment.—*Treatment* seems to be of little avail.

Picro-nitrate of potassium has been advised.

NOCTURNAL INCONTINENCE OF URINE IN CHILDREN.

Causes.—1. *Acidity of the urine.*

2. *Irritability of the bladder.*

3. An *adherent prepuce*, or thread worms in the bowel or vagina.

4. A *weak state of the sphincter muscle.*

Treatment.—1. To *lessen the acidity of the urine* by giving bicarbonate of potash.

2. To *lessen the amount of urine formed* by giving the child only a small quantity of fluid in the evenings.

3. To *lessen the irritability of the bladder* by belladonna and bromide of potassium.

4. To *relieve an adherent prepuce* or *remove worms.*

5. To *strengthen the sphincter muscle* by strychnine and cantharides.

MORBID CONDITIONS OF THE URINE AND THEIR SIGNIFICANCE.

Loss of Transparency may be due to the presence in the urine of 1. Mucus. 2. Urates. 3. Phosphates. 4. Pus. 5. Blood. 6. Bacteria.

1. *Mucus* in the urine, if in excessive quantity, shows some irritative or inflammatory condition of the bladder.

2. Urine containing an excess of *urates* is clear when passed, but the urates are deposited as a pinkish sediment on cooling. Their presence is of very little clinical significance, but calculi may be formed from them.

3. The presence of *phosphates* in excess shows some digestive disturbance and may lead to the formation of a phosphatic calculus. The condition leading to their excessive formation is best treated by the mineral acids and nux vomica.

4. *Pus* in the urine may be due to urethritis, cystitis, pyetitis, nephritis or to the bursting of an abscess into the urinary passages.

5. *Bloody* urine has already been considered.

6. *Bacteria* always develops in urine a few hours after it is passed, unless it is sterilized, and they may develop in the bladder before the urine is passed if an unclean catheter has been passed.

The Color of the Urine depends upon (1) the *quantity* passed; (2) the *coloring matter* and other substances which it contains; it is rendered dark by bile and smoky by blood. It is high colored in fever cases, but the color under these circumstances is of little significance.

The Quantity of Urine passed depends in a general way upon (1) the amount of fluid taken into the body; (2) the blood pressure. It is nearly always greatly increased in cases of *diabetes mellitus*.

The Specific Gravity is usually inversely in proportion to the quantity passed, *except in cases of diabetes*, when the amount is large and the sp. gr. high, from 1025 to 1040.

Reaction of the Urine.—The urine may become alkaline *from fermentation* or it may be *excessively acid* from an excess of uric acid.

Urinary Deposits.—The most common urinary deposits are (1) urates of soda and ammonia; (2) earthy phosphates; (3) oxalate of lime, and (4) uric acid.

The significance of the *urates* and phosphates has already been stated.

Oxalate of lime appears in the urine as a result of imperfect metabolism or from the use of certain articles of food (sorrel). If the deposit persists it is liable to form a calculus.

Uric acid is often found in the urine in excess; it is nearly related to the *gouty diathesis*, and is due to the inadequacy of the liver.

The following table for facilitating the examination of urinary deposits by chemical means and by the microscope is a modification of that of Bowman :

1st. Chemical Examination—

1. The sediment dissolves when warmed . . . *Urates*
2. Not soluble when warmed, but soluble in acetic acid *Earthy phosphates*
3. Insoluble in acetic acid, but soluble in dilute hydrochloric acid *Oxalate of lime*
4. Insoluble in dilute hydrochloric acid, but purple with nitric acid and ammonia . . *Uric acid*
5. Soluble in ammonia the solution leaving on evaporation hexagonal crystals . . *Cystine*
6. Milky appearance rendered clear by ether *Chylous urine*

2nd. Microscopical Examination—

1. If the deposit is crystalline—
 - (1) Lozenge-shaped, rhomboidal or stellate crystals *Uric acid*
 - (2) Three-sided prisms with beveled edges and truncated ends, or feathery, fern-like crystals *Triple phosphates*
 - (3) Octahedral (envelope) or dumb-bell crystals *Oxalate of lime*
 - (4) Rosette-like tables *Cystine*
 - (5) Needle-shaped crystals, grouped in bundles or globular masses . . . *Tyrosine*
2. If amorphous or rounded particles—
 - (1) Soluble when warmed *Urates of soda*
 - (2) Soluble in acetic acid *Phosphate of lime*
 - (3) Yellowish grains, often spicular . . . *Urate of ammonia*
 - (4) Round globules, with dark edges . . *Fat*
 - (5) Dark globules resembling fat . . . *Leucine*
 - (6) White and milky *Chyle*
3. If organized particles—
 - (1) Granular corpuscles in stringy aggregation *Muco-pus.*
 - (2) Detached granular corpuscles . . . *Pus*
 - (3) Irregularly-shaped scales *Epithelium*

The significance of these various deposits has already been stated.

Tube casts were described in an earlier part of this chapter.

CHAPTER VII.

DISEASES OF THE ORGANS OF LOCOMOTION.

ACUTE ARTICULAR RHEUMATISM.

(Inflammatory Rheumatism.)

Definition and Frequency.—An acute general disease characterized by inflammation of one or more of the larger joints and probably due to the presence of lactic acid in the blood.

It is of very common occurrence.

Causes.—1. The *essential cause* is probably the presence of *lactic acid* in the blood; probably this results from the action of a germ on albuminoid bodies.

2. The disease is most common in *temperate climates* and especially after exposure to *cold* and *dampness*.

3. *Age*. It is most common between the ages of fifteen and forty, and—

4. *Heredity* seems to play a certain part in its production.

5. *Impairment of the general health* seems to be a predisposing cause.

Morbid Anatomy.—1. The *blood* contains lactic acid and fibrin factors in excess (?).

2. The *joints* are swollen, the synovial membranes somewhat reddened and there is a serous effusion or exudate into the affected joints.

Symptoms.—A. *Premonitory*. 1. *Sore throat* (tonsilitis) is a frequent precursor of rheumatic fever.

2. Some *digestive disturbances*, such as cholera morbus, may also occur.

B Symptoms of the *developed attack*.

1. The *joints*. One or more of the large joints are involved and frequently the disease may suddenly disappear from one joint and appear in another. They are swollen, excessively tender and painful on movement and usually somewhat red.

2. The *temperature* is elevated usually from 103° to 105° , but it may reach 110° .

3. *Circulatory*. The pulse is quick and often jerky, probably from the effect of the lactic acid in the blood on the heart.

4. *Cutaneous*. *Acid sweats* occur in almost all cases and at frequent intervals.

5. The *urine* is apt to be excessively acid and rarely it contains albumin.

6. *Digestive.* *Acid dyspepsia* is a frequent concomitant; constipation is usually present.

7. *Nervous.* Besides the pain in the joints there may be headache and occasionally violent delirium.

Complications.—1. *Cardiac.* Endocarditis occurs in about one-third of the cases and pericarditis nearly as often. They are doubtless due to the morbid matters in the blood.

2. *Nervous.* Delirium has already been mentioned, but it is sometimes so prominent as to be a serious complication (cerebral rheumatism.)

Sequelæ.—1. *Valvular disease of the heart* is the most common and by far the most serious sequel.

2. *Chorea* occurs as a sequel in many cases.

3. *Chronic inflammation* of the joints rarely results.

Diagnosis.—1. From *pyæmia* it is distinguished by the history of the case and the more severe constitutional disturbance in pyæmia.

2. From *simple synovitis* by the fact that in synovitis but one joint is usually involved and there is no acid sweat.

Prognosis.—The disease is rarely fatal, the mortality being only about 3 per cent.; it is dangerous from the results (valvular disease).

Duration.—The usual *duration* is from three to six weeks, but mild cases may terminate in a day or two.

Treatment.—A. *Hygienic.* The room should be large and airy; the clothing should be warm (flannel); the diet should be light and unstimulating.

B. *Medicinal.* The indications are—

1. To *relieve pain* and *shorten the disease*.

2. To *prevent cardiac complications*.

1. To *relieve pain* *salicylic, acid* or *salicylate of soda* is by far the best drug. It rarely shortens the course of the disease and it does not prevent cardiac complications. *Salol* has a very similar effect. *Phenacetine* is less useful. *Oil of wintergreen* has the same effect as salicylic acid.

Morphia or codeia may be given, but are rarely necessary.

Locally ice bags, poultices, chloroform and aconite and blisters have been used to relieve pain; they are of doubtful value. The limb may be enveloped in cotton batting with advantage.

2. To *prevent cardiac complications* the alkalis, such as bi-carbonate of potassium, are of undoubted value.

Relapses are common, and rest in bed with persistent use of salicylate of soda is advisable for a time after apparent recovery.

CHRONIC ARTICULAR RHEUMATISM.

Causes.—This disease is far more common in *elderly people* than in young ones; it seems to be *hereditary* and exposure to cold and dampness is an exciting cause; *rare weather* will bring on an attack in persons liable to it.

Morbid Anatomy.—The *joints* are somewhat swollen and the synovial sack and sheathes of the tendons are thickened.

Symptoms.—The *joints* are more or less stiff and painful; the pain being usually dull and aching in character. The knees, ankles and wrists are usually involved.

Diagnosis.—It is diagnosed from rheumatic arthritis by the fact that the large joints are almost exclusively involved and the disease is far less severe than rheumatoid arthritis.

Prognosis.—The *prognosis* as to life is good; as to perfect recovery is bad.

Treatment.—The *indications* are—

1. To *relieve pain*.
 2. To *relieve stiffness and reduce inflammation*.
 3. To *build up the general health*.
1. To *relieve pain* counter-irritants, and anodyne applications are useful.
 2. *Stiffness and inflammation* are benefitted by massage, electricity and iodide of potassium.
 3. *Cod liver oil and iron* are often useful.

ARTHRITIS DEFORMANS.

(Rheumatoid Arthritis.)

Definition.—A chronic and usually progressive disease affecting usually the smaller joints and causing deformity and loss of motor power.

Causes.—1. *Age and sex*. Women after middle life are usually affected.

2. *Menstrual disturbances* (Ord) seem to have some influence on its production.

3. *Exposure and over-work* are said to be causes.

Morbid Anatomy.—The *articular cartilages* are ulcerated and finally destroyed; the *synovial fringes* greatly enlarged and thickened; the *ends of the bones* “*eburnated*” and sometimes connected

by fibrous bands and often plates of bone form on the synovial sack surrounding the joints. The *adjacent muscles* waste.

Symptoms.—1. The *smaller joints* are usually involved, especially those of the fingers, but the knee, shoulder and other joints may also be affected. The change in the joints from ulceration of the cartilages and disease of the bones leads to *deformity*.

2. *Pain* is very great whenever any movement is attempted and often without movement.

3. *Anæmia* is often present.

Diagnosis.—It is distinguished from *gout* by the absence of the attacks which are so characteristic of gout and of regular *tophi* in the joints.

From *rheumatism* by the fact that the smaller joints are chiefly affected and there is much greater deformity. (Many physicians however class arthritis deformans and chronic rheumatism together).

Prognosis.—The disease is usually progressive and incurable; but recoveries sometimes occur. In the most severe cases there seems to be no tendency to shorten life.

Treatment.—A *warm, dry climate* is very beneficial.

Cod liver oil and *iodide of iron* to improve the general health are useful and also arsenic.

Hot baths and especially *hot douches* give good results sometimes.

Massage and *electricity* are indicated if they do not cause very severe pain.

MYALGIA.

(Muscular Rheumatism.)

Definition.—An affection of certain muscles or groups of muscles characterized by pain and stiffness.

Causes.—1. *Exposure to cold and dampness* is by far the most common cause.

2. *Strains* will often cause an attack, such as lumbago.

3. *Rheumatism* and *gout* and malaria are said to be causes.

Morbid Anatomy.—There is no characteristic morbid change.

Symptoms.—*Pain*, especially on movement, and *stiffness* of certain muscles or groups of muscles, causing—(1) Torticollis or wry neck; (2) lumbago; (3) myalgia of the muscles of the arms and shoulders, &c.

Diagnosis.—It is distinguished from (1) *disease of the spine* by the absence of any disturbance about the vertebrae; (2) *locomotor ataxia* by the sudden and transitory character of the lightning pains in this disease; (3) *pleurisy* by the absence of fever and pleuritic friction sounds.

Prognosis.—The *prognosis* is always good and the *duration* usually only a few days.

Treatment.—1. To *relieve pain* by (1) hot applications; (2) cupping; (3) phenacetine; (4) opiates; (5) quinine; (7) electricity.
2. To *relieve stiffness* by electricity and massage.

GOUT.

Definition.—A disease characterized by an excess of uric acid in the blood and usually by the presence in old cases of deposits of urates (tophi) in the smaller joints.

Causes.—1. *Hereditv.* In a large proportion of cases the disease is distinctly hereditary; no explanation of this fact can be given.

2. *Age.* It usually occurs first, especially the *acute* form, between thirty-five and forty, probably because the causes to be mentioned next are in more active operation at this period of life.

3. *Retention in the body of nitrogenous waste matters* from (1) over-eating; (2) defective oxidation; (3) inadequacy of the kidneys.

Nitrogenous food is chiefly burnt off to form urea in the *liver* and if this organ cannot do its work properly, *uric acid* instead of urea is formed. The formation of urea is a process of oxidation, and the more sedentary the life the less active is this process of oxidation. Finally nitrogenous waste is removed chiefly by the kidneys, and uric acid and urate of soda are comparatively insoluble, so if the kidneys are not doing their work properly these substances accumulate in the blood and cause gout.

4. *Chronic lead poisoning*, even if slight, renders a person far more liable to gout probably by interfering in some way with the action of the kidneys.

Morbid Anatomy.—1. The *blood* contains uric acid in excess.

2. The *joints*, especially the smaller ones of the fingers and toes, are often enlarged and the cartilages are infiltrated and covered with urate of soda, which in many cases of old standing, forms chalk stones (*tophi*) around the joints also.

3. The *kidneys* are often small, their connective tissue is increased and urates are found in the urinary tubules.

4. The *arteries* show arterio-capillary fibrosis, and the heart is enlarged in consequence.

The change in the kidneys and arteries is probably due to the irritation and inflammation set up by the uric acid in the blood.

Symptoms.—A. *Prodromic.* The symptoms which sometimes (not always) precede an *acute* attack of gout are, irritability of temper, mental depression, flatulence or other dyspeptic symptoms and the presence of large quantities of lithates in the urine.

B. *During an attack of acute gout.*

1. The *onset* is sudden, the attack usually occurring about 2 o'clock in the morning

2. *Nervous.* Pain, nearly always in one big toe joint, is by far the most conspicuous nervous symptom. There is great tenderness as well.

3. The *joint* affected is swollen, red and exquisitely sensitive.

4. The *temperature* is elevated 102° to 103° .

5. The *pulse* is full and strong and considerably quickened.

6. The *digestive* symptoms consist in flatulence, heartburn and eructations of gas.

7. The *urine* shows a great *diminution* in the amount of *uric acid* discharged.

The *duration* of such an attack is two or three hours generally, but the joint is left swollen and tender and the attacks are apt to recur every night for ten days or two weeks.

C. *Of chronic gout.*

1. *Tophi*, or deposits of urates, in and around the joints—("chalk stones").

2. *Cutaneous.* Eczema is a common symptom; it is probably due to the irritation of the skin by the uric acid in the course of its elimination by it.

3. *Digestive.* *Gastro-enteritis* is an occasional symptom or *form* of chronic gout.

4. *Nervous.* *Vertigo*, *numbness*, *headache*, *neuralgia* and other nervous symptoms are common.

5. *Pulmonary.* Bronchitis, according to English physicians, is common.

Sequelæ.—1. *Circulatory.* Arterio-capillary fibrosis and hypertrophy of the heart (see Morbid Anatomy).

2. *Pulmonary.* *Asthma* is common in the course of gout rather than as a sequel; it is probably due to the irritation of the nerve centers, and possibly of the bronchial tubes themselves, by the uric acid retained in the blood.

3. *Digestive.* *Gastro-intestinal catarrh* and *jaundice*. The former is probably caused by the irritation of the uric acid and the jaundice results from the catarrh.

Diagnosis.—It is distinguished from *rheumatism* by (1) the history of acute attacks; (2) the tophi around the joints; (3) the involvement, as a rule, of the *smaller* joints.

Prognosis.—The *prognosis* is based on (1) the *form* of the disease—gastro-intestinal attacks in gout are very dangerous; (2) on the *age* at which it first occurs—the younger the person the worse the prognosis; (3) the complications; (4) the number of joints involved.

Treatment.—The indications are—

1. To *increase oxidation* and thus to have more urea and less uric acid formed.

2. To *increase the functional activity of the liver* and, at the same time, to *lessen the work of the liver*.

3. To *increase the functional activity of the kidneys* and thus eliminate uric acid.

Active exercise is beneficial because it increases the oxidation and the work of the skin which acts vicariously for the kidneys.

A *warm climate* and *warm clothing* promote the action of the skin and lessen the work of the kidneys.

The *diet* is of the greatest importance; it should be *very scant*, especially in nitrogenous food and rich or highly seasoned articles; vegetables, especially those containing little starch, milk, bread and small quantities of meat are advisable. Sweet wines, and most liquors, are especially injurious; if stimulants are absolutely essential, gin and whiskey are least hurtful. Large quantities of water are useful to wash out the kidneys. Urate of lithia or potash is more soluble than either uric acid or urate of soda, and hence these *alkalies* are of service. *Opium* and *colchicum* may be used to relieve pain.

Cold baths to the foot during a paroxysm are not advisable; warmth is often of service. During an acute attack the foot should be raised, the big toe joint painted with morphia solution or chloral-camphor and then wrapped in flannel.

RICKETS.

(Rachitis.)

Definition.—A disease of infancy and early childhood characterized by a defective deposit of lime salts in the bones.

Causes.—1. *Bad hygienic conditions*, especially over-crowding and improper food.

2. Probably *bad health of the parents*, especially of the mother.

3. *Age*. It nearly always occurs within the first eighteen months or two years of life.

Morbid Anatomy.—1. The *bones* are abnormally soft, the epiphyses are enlarged and the bones often bent.

2. The *skull* presents striking features; the fontanelles are large, the sutures unclosed and the edges of the bones often greatly thickened.

3. The *muscles* are thin and flabby.

Symptoms.—1. *Digestive*. The *abdomen* is usually very large, and flatulence and whitish pasty actions are frequent. The *teeth* are cut very late.

2. The most important *nervous* symptom is laryngismus stridulus, but convulsions are not uncommon.

3. The *limbs* are small, often bent, and the ends of the long bones are enlarged.

4. The *general* symptoms are weakness and pallor.

Diagnosis.—The *diagnosis* is based on the appearance of the limbs and skull.

Prognosis.—The *prognosis* depends upon the possibility of removing the cause.

Treatment.—A. *Hygienic*. Fresh air and proper food—*milk*—are of the first importance.

B. *Medicinal*. Cod liver oil, iodide of iron and lacto-phosphate of lime are the agents which have given best results.

OSTEO-MALACIA.

(Mollities ossium. Malacosteon.)

Osteo-malacia is an acquired softening of bones occurring in adult life. It is common in some parts of Europe but rare in America.

The *causes* are obscure; it occurs chiefly in women, especially during pregnancy and about the age of thirty-five.

The *morbid anatomy* consists, primarily, in softening of the bones, and secondarily, in certain *deformities* and fractures resulting

therefrom. Malacosteon pelvis is the most important deformity from a practical standpoint.

The *symptoms* are at first, pain in the lower part of the back, which is not characteristic, but which is followed by changes in the bones.

The *diagnosis* is plain.

The *prognosis* is bad and the disease progressive.

The only *treatment* which has been of any avail is the removal of the ovaries and tubes.

CHRONIC GENERAL DISEASES.

DIABETES MELLITUS.

Definition.—A disease characterized by the presence of *sugar in the urine*. If temporary in character it is called *glycosuria*.

Morbid Anatomy.—1. The *blood* contains sugar, is sometimes thicker than normal and coagulates badly.

2. The *liver* is hyperæmic.

3. The *lungs* nearly always contain tubercles, and gangrene is not uncommon.

4. The *skin* is dry and boils and carbuncles occur very frequently.

5. The *pancreas* has been found diseased (atrophied usually) in a number of cases.

Causes.—1. *Age and sex*. Men in early adult life are most subject to the disease.

2. *Nervous troubles*, tumors in the 4th ventricle, emotional disturbance, or injuries about the head sometimes cause it.

3. *Heredity*. Diabetes occurs with especial frequency in families which are liable to nervous troubles.

Injudicious eating will add greatly to the severity of the disease.

Symptoms.—1. The *onset* is insidious.

2. *Urinary*. The *amount* of urine is nearly always greatly increased; the specific gravity is high—from 1025 to 1040; and *sugar* is *present* in greater or less amount; as much as a pound and a half or two pounds may be discharged per day.

3. *Digestive*. *Thirst* is excessive, and often there is a ravenous appetite; constipation is present, as a rule.

4. *Cutaneous*. The dryness of the skin and the frequent occurrence of boils have been mentioned.

5. *Nervous*. *Neuralgia*, especially *sciatica*, is of common occurrence; headache and lassitude are also common, and in the late stages *coma* may come on suddenly or be preceded for a short time by delirium. The *coma* ("diabetic coma") is probably due to the presence of β -oxy-butyric acid in the blood.

6. The *temperature* is often below normal, the pulse weak and the respirations slow.

Diagnosis.—The *diagnosis* is based on the presence of sugar in the urine. Trommer's test for sugar is as follows: Add to the suspected urine enough liquor potassæ to render it distinctly alkaline, then add a few drops of a solution of cupric sulphate and boil; if sugar be present a red precipitate will be formed.

Prognosis.—The *prognosis* is serious and the disease usually progressive, but life may be prolonged for months or years by prudence as to diet.

Complications.—1. *Pulmonary*. Phthisis is a very frequent complication; pneumonia and pulmonary gangrene also occur.

2. *Cutaneous*. The *boils* and carbuncles already mentioned.

3. *Urinary*. *Suppression of urine* and *uræmia* may occur in the late stages.

4. *Ocular*. Cataract occurs with comparative frequency in diabetics.

Treatment.—A. *Dietetic*. All *saccharine food* should be avoided and *nearly all starchy food*.

The following articles of food may be used.

1. *Meat* of all kinds, except liver; fish of all kinds, except oysters.

2. *Bread* made of bran, almond flour or soya beans. (Most of the so-called diabetic flour contains starch in large quantity).

3. *Butter*, cream and fat of any kind.

4. *Vegetables*. Green vegetables, such as spinach, salads, turnip tops, etc.

5. *Liquids*. Water, buttermilk in moderation, tea, coffee and acid wines.

Saccharine should be used in place of sugar; one grain will sweeten a cup of tea or coffee.

The following articles should be avoided.

1. All *saccharine* and *starchy food*, such as ordinary bread, potatoes and white vegetables, and also beets, turnips, carrots, &c., which contain sugar.

2. All sweet wines and also chocolate.

B. *Medicinal*. The indications are to lessen the amount of sugar discharged and relieve thirst. For this purpose the following

remedies are useful—(1) codeia (or opium in some form); (2) bromide of arsenic; (3) carbolic and salicylic acids; (4) antipyrine; (5) lithia water.

DIABETES INSIPIDUS.

(Polyuria.)

Definition.—A disease characterized by the persistent discharge of large quantities of limpid urine.

Causes.—1. Injuries and diseases of the nervous system.
2. Emotional disturbance, if serious and prolonged.

Morbid Anatomy.—Often there is none. Sometimes disease of the floor of the 4th ventricle exists.

Symptoms.—1. *Urinary.* The quantity of urine is enormously increased; the *specific gravity* is low—1004 or 1005.

2. *Digestive.* *Thirst* is excessive and constipation usually exists.

3. *Cutaneous.* The skin is dry and harsh.

Diagnosis.—The diagnosis is based on the urinary symptoms, and especially on the persistence of these symptoms and the absence of organic kidney disease, &c.

Prognosis and Duration.—Recovery is rare, but patients may live for years with this disease.

Treatment.—A. *Hygienic and Dietetic.* Care should be taken to keep up the general health. No change of diet is necessary except to diminish the amount of liquid ingested as far as possible.

B. *Medicinal.*—The indication is to lessen the quantity of urine discharged and to allay thirst. The following drugs have been found useful (1) valerian; (2) ergot; (3) salicylate of soda.

ANÆMIA AND CHLOROSIS.

Definition and Synonyms.—By *anæmia* is meant a diminution in the number of red corpuscles in the blood. It is sometimes called oligocythæmia.

Chlorosis is a form of anæmia usually occurring in girls about the age of puberty without obvious causes.

Causes.—Anæmia is due to—

1. Discharges of blood and pus.
2. The influence of certain chronic diseases, such as phthisis, cancer and Bright's.

3. Malnutrition, from narrowing of œsophagus, &c.

The causes of chlorosis are unknown.

Morbid Anatomy.—1. The *blood* is more watery than normal, the number of red corpuscles being only 600,000 or 800,000 per cubic millimeter, instead of 5,000,000. The red corpuscles themselves are often changed, some being larger than natural, many smaller (microcytes) and some elongated or comet-shaped (poikilocytes).

2. The *heart, arteries* and muscular tissue generally, in severe cases, show some fatty degeneration, and the heart is often enlarged from loss of muscular tone and consequent distension.

3. The *skin* is pale; in chlorosis it sometimes has a greenish tinge.

Symptoms.—A. The symptoms of *acute* anæmia from sudden loss of a large quantity of blood are, pallor, faintness, dyspnœa, yawning, restlessness and feeble pulse.

B. The symptoms of chronic anæmia and chlorosis are—

1. *Cutaneous*. Pallor is the most marked.

2. *Circulatory*. The *pulse* is soft and compressible, and becomes very rapid on slight exertion.

A *murmur*, often harsh in character, but often blowing, is heard over the heart, most clearly at the 2nd left costo-sternal interspace close to the sternum; it is propagated a little way to the left, just below the clavicle. A blowing sound is heard also over the large veins in the neck; the cause of these sounds is unknown. Palpitation of the heart is common, especially on exertion.

3. *Respiratory*. There is dyspnœa on exertion, because the red corpuscles (the oxygen carriers) are diminished in number.

4. *Nervous*. Headache and hysterical symptoms are common, and are probably due to defective nutrition of the nerve centers.

5. *Digestive*. Indigestion, which is of frequent occurrence, is due to the want of a proper blood supply to the gastric tubules and the muscular walls of the stomach and bowels.

6. *Menstrual*. As a rule, the menstrual discharge is diminished, or, more frequently, suspended in cases of anæmia.

7. *General*. There is no loss of weight, as a rule, in chlorosis.

Diagnosis.—The *diagnosis* of anæmia is perfectly easy.

Chlorosis is distinguished from simple anæmia by the absence of any apparent cause in the case of the former.

Prognosis.—The prognosis of simple anæmia depends upon the cause.

In the case of chlorosis the prognosis is always good, but relapses are liable to occur.

Complications.—The complications of chlorosis are:

1. Ulcer of the stomach.
2. Pulmonary phthisis; it is questionable whether the anæmia may not be secondary in these cases to the phthisis, which could not at first be detected.

Treatment.—The indication is to increase the number red corpuscles in the blood.

A. *Hygiene and diet.* Exercise in the fresh air is very important, because more oxygen is obtained, the circulation is rendered more free and nutrition is increased.

The diet should be nutritious and digestible, meat being of especial value. Ale, beer and wine are often of service.

Cheerful company and change of air and scene often “work wonders” in chlorosis.

B. *Medicinal.* Iron is the great remedy in these cases, but it acts much better, usually, if given in combination with—

2. *Potash.* Blaud’s pills contain iron and potash.
3. *Arsenic* is useful especially in chlorosis.
4. *Cod-liver oil* is of service if there is much loss of flesh.

PROGRESSIVE PERNICIOUS ANÆMIA.

(Pernicious anæmia. Idiopathic anæmia.)

Definition. A form of anæmia progressive in course and usually terminating fatally.

Causes.—The causes are practically unknown. It is more common in *women* than in men, and seems to be often connected with pregnancy.

Morbid Anatomy.—1. The *blood* changes are like those of simple anæmia.

2. The *marrow* of the bones in many cases is red, because the fat cells have disappeared, and red corpuscles, usually large and nucleated are found in great numbers.

3. The *gastric tubules*, according to Fenwick, are often atrophied.

4. The *heart and blood vessels* are usually fatty.

Symptoms.—1. *General.* The *onset* is gradual; there is no emaciation, as a rule, but there is a progressive loss of strength.

2. *Circulatory.* The pulse is soft and compressible and in the late stages rapid and very feeble.

Hemorrhages from the nose or other parts are of frequent occurrence and may be profuse.

3. *Ocular.* On ophthalmoscopic examination there are found to be hemorrhages into the retina in the majority of cases and vision is more or less impaired in consequence.

4. *Digestive.* Nausea and vomiting are common and often troublesome symptoms.

5. *Temperature.* There are irregular rises of temperature, 102° to 103° in the late stages.

6. Delirium may occur also at this period.

Diagnosis.—1. From *simple anæmia* it is distinguished by its progressive course and the occurrence of hemorrhages.

2. From *leucocythæmia* by the absence of any increase of white corpuscles in the blood.

3. From *Hodgkin's disease* by the absence of glandular swellings.

Prognosis.—The *prognosis* is extremely bad, and the great majority of cases terminate fatally.

Treatment.—*Arsenic* is the only remedy which has given any really satisfactory results in this disease. The dose of Fowler's solution should gradually be increased up to fifteen or eighteen drops three times a day.

Iron, zinc, phosphorous, quinine and various other remedies have been tried without benefit.

LEUKÆMIA.

(Leucocythæmia.)

Definition.—A disease characterized by a great increase in the number of white corpuscles in the blood and by enlargement of the spleen or lymph glands, or both.

Causes.—The *causes* are practically unknown.

Morbid Anatomy.—1. The *blood* shows a great increase in the number of white corpuscles, the proportion to the red being 1 to 60, 1 to 20, or there may even be more white than red (normally the proportion is 1 white to about 300 red.)

2. The *spleen* is usually enlarged to a greater or less extent, it may be very greatly, the enlargement being usually due to a simple



increase in its normal constituents; but in old cases the amount of connective tissue is increased.

3. The *lymph glands* frequently undergo a similar change.

Symptoms.—1. *Circulatory.* The changes in the *blood* have already been described. The *pulse* is weak.

The *spleen* and *lymph glands* are enlarged.

Hemorrhages sometimes occur.

2. The *skin* is pale and occasionally hemorrhages occur into it.

3. The *temperature* is usually normal till the latter stages of the disease, and then there are irregular elevations, 102° or 102.5° .

4. The *general* symptoms are loss of strength and vigor; there is usually no emaciation.

Diagnosis.—The *diagnosis* is simple and is based on a microscopic examination of the blood.

Prognosis.—The *prognosis* is extremely bad; death usually occurs from exhaustion in from three months to three years.

Treatment.—*Arsenic* has given better results than other remedy.

Oxygen inhalations have been tried recently with good results.

Quinine and iron are useless.

Transfusion of blood has been tried without success.

HODGKIN'S DISEASE.

(Psuedo-leukæmia. Lymphadenoma.)

Definition.—A disease characterized by progressive enlargement of the glands in different parts of the body, without increase of white blood corpuscles and by great anæmia.

Causes.—The *causes* are unknown.

Morbid Anatomy.—1. The lymphatic glands in the neck, axilla, groin, thorax and abdomen are greatly enlarged, hard and occasionally caseous. Their connective tissue is increased.

2. The *spleen* undergoes a similar change.

3. Adenoid growths are found in the liver and various other parts.

Symptoms.—1. *General.* Progressive loss of strength and sometimes of flesh.

2. *Glandular.* The glands are enlarged as already stated.

3. *Circulatory*. The pulse is soft and feeble and hemorrhages sometimes occur.

4. *Temperature*. There are irregular elevations of temperature in the late stages.

Diagnosis.—The *diagnosis* is based on (1) the glandular swelling; (2) the absence of any increase of white corpuscles in the blood.

Prognosis.—The disease usually terminates in death; the duration is about a year.

Treatment.—*Arsenic* internally and injected into the growths is said to have given good results. *Phosphorus* is advised. Cod-liver oil, quinine and iron are useless.

Excision of the glands has been tried; it is not usually of service.

ADDISON'S DISEASE.

Definition.—A chronic disease, characterized by a bronze color of the skin and usually by disease of the supra-renal capsules.

Causes.—1. *Age and sex*. It is most common in young men or men in middle life.

2. The bacilli *tuberculosis* are considered the cause by many who hold that the disease is tuberculosis of the supra-renal capsules.

Morbid Anatomy.—1. The *skin* is of a bronze color, especially in the face, hands, and places which are subjected to pressure and in situations where there is naturally much pigment.

2. The *supra-renal capsules* are usually diseased; they may be caseous or have undergone "fibroid degeneration."

3. The *spleen* is somewhat enlarged in many cases and the *lymph glands* and *Peyer's patches* also.

4. The *semi-lunar ganglion* shows degeneration and pigmentation of its cells.

Symptoms.—1. *General*. The *onset* is gradual; there is no emaciation, but a gradual loss of strength, with intervals, however, of apparent improvement.

2. The *nervous* symptoms are depression, lassitude and hypochondria.

3. The *digestive* disturbances are nausea and vomiting and there is loss of appetite.

4. *Circulatory.* The pulse is weak and compressible and faintness may occur on exertion.

5. The *temperature* is usually normal, but there may be occasional elevations.

6. The *bronze color of the skin* is the most striking symptom.

Diagnosis.—It is distinguished from *jaundice* by the absence of discoloration of the sclerotic.

In one case Koch's lymph has been used successfully for diagnosis.

Prognosis.—The *prognosis* is bad, and the duration usually about eighteen months.

Treatment.—Thus far treatment has been useless.

SCURVY OR SCORBUTUS.

Definition.—A disease characterized by swelling of the gums and by hemorrhagic extravasations in different parts of the body.

Causes.—1. *Bad hygienic* surroundings, probably, predispose to it.

2. *Improper food*, especially the *want of fresh vegetables*, is the exciting cause.

Morbid Anatomy.—1. The red corpuscles are diminished in number and hemorrhagic extravasations occur in the skin and sometimes in the muscles. The heart is softened.

2. The *spleen* is enlarged and softened.

3. The *gums* are swollen and bleed readily and the teeth are loosened.

Symptoms.—1. *Cutaneous.* The skin is pale and bloated and the seat of large or small hemorrhagic extravasations.

2. *Circulatory.* The *pulse* is slow, unless quickened by exertion, and is also very weak and compressible.

3. The *temperature* is usually below normal.

4. *Nervous.* There is pain in the back and limbs, and the latter are often hard and swollen from extravasation of blood. There is also great depression and loss of mental power.

5. The *teeth* are loosened and the *gums swollen*.

Diagnosis.—1. From *mercurial stomatitis* it is distinguished by the presence of hemorrhagic extravasations in scurvy and by the history of the case.

2. From *purpura* by the absence of any serious involvement of the gums in the latter.

Prognosis.—The *prognosis* is good.

Treatment.—A. *Dictetic*. Fresh vegetables and lime or lemon juice are both preventive and curative.

B. *Medicinal*. The medicinal treatment consists in building up the general health by tonics, such as quinine, iron, quassia and the other agents of this class, and by the use of *chlorate of potash*, which should be taken internally and used as a mouth wash. Solutions of carbolic acid, permanganate of potash and boracic acid are also useful as mouth washes.

PURPURA.

Definition.—A disease characterized by either extravasations of blood in the skin and mucous membranes or by hemorrhages from various mucous membranes.

Causes.—The causes are practically unknown. The disease sometimes follows *rheumatism* and *diphtheria*, but, in the great majority of cases, no cause can be found.

Morbid Anatomy.—*Extravasations* of blood in the skin and mucous membranes are the only morbid appearances in many cases. Small extravasations are but little elevated, but large ones, two or three inches in diameter, are elevated.

Symptoms.—A. *Prodromic*. Occasionally there is a feeling of lassitude and depression before the purpuric spots appear; often there are no prodromata.

B. After the spots appear they, or *hemorrhages*, are usually the only symptoms.

Pallor and other symptoms of anæmia result from loss of blood.

Pain may occur from distension of the tissues at the seat of extravasation.

Diagnosis.—It is distinguished from *scurvy* by the absence of swelling of the gums and of serious constitutional disturbance in the early stages.

Prognosis.—In the *simple form* the prognosis is good; in the hemorrhagic form it is serious. The duration in simple cases is rarely more than a few weeks; in the hemorrhagic form it may be fatal in a few weeks or may last much longer.

Treatment.—A. *Hygienic*. Rest, fresh air and nutritious food are important.

B. *Medicinal*. The medicines most used are—

1. *Iron*, to improve the character of the blood and the nutrition of the walls of the vessels.

2. *Sulphuric acid, ergot, turpentine and gallic acid* to control hemorrhage. Turpentine and gallic acid are especially useful when there is hæmaturia.

HÆMOPHILIA.

Definition.—A constitutional tendency to bleed profusely from slight injuries. Persons having such tendency are called “bleeders.”

Causes.—1. *Sex.* Males are more liable to the affection than females; the tendency usually develops in the first few years of life.

2. *Heredity*; the transmission is through the mother and not the father.

Morbid Anatomy.—There is no characteristic morbid anatomy. Extravasations of blood in the joints, especially the knee joints, is frequent, however.

Symptoms.—*Hemorrhage*, from slight injuries, which is profuse and uncontrollable.

Prognosis.—The *prognosis* of such cases is always serious.

Treatment.—The *treatment* is purely preventive.

ALCOHOLISM.

Divisions.—1. Acute alcoholism; (1) Drunkenness; (2) Delirium tremens.

2. Chronic alcoholism.

Drunkenness will not be considered here.

Causes of delirium tremens.—1. Excessive drinking.

2. A prolonged debauch.

3. *Injuries and certain acute diseases*, such as pneumonia, will bring on an attack in persons who have been drinking heavily.

Morbid Anatomy.—A. *Acute alcoholism.*—1. The *stomach and duodenum* are greatly inflamed.

2. The *brain*, and especially the membranes surrounding it, are congested.

3. The *liver, kidneys, lungs and bronchi* are hyperæmic.

B. *Chronic alcoholism*.—1 The *stomach* shows the usual evidences of chronic inflammation (q. v.).

2. The *liver* and *kidneys* are often cirrhotic.

3. The *lungs* and *bronchi*—especially the latter—are inflamed from elimination of alcohol.

4. The *brain* shows decided changes; the *membranes* are thickened and opaque and the *convolutions* show an increase of connective tissue.

5. The *arteries* are usually *atheromatous* from the action of the alcohol and nitrogenous waste which is retained in the blood.

6. The *muscular* tissue generally is fatty.

Symptoms.—A. *Delirium tremens*.—1. *Nervous*. Excitement, with wild delirium at night, first occurs; in the day, for one or two days, the patient has little or no delirium, but after the second or third day the delirium persists, and there are hallucinations usually of a painful or unpleasant character. *Sleeplessness* always exists.

2. *Digestive*. There is complete loss of appetite, nausea and vomiting, as a rule.

3. *Circulatory*. The *pulse* is rapid, soft and compressible, and prostration is often extreme.

An attack of delirium tremens usually lasts three or four days and ends with long and quiet sleep.

B. *Chronic alcoholism*.—1. *Nervous*. Tremor of the hands, which is most marked in the morning, want of decision of character and gradual enfeeblement of the intellect, are the most important nervous symptoms.

2. *Digestive*. *Vomiting*, especially in the morning, and loss of appetite are nearly always present; in many cases there are evidences of cirrhosis of the liver.

3. *Circulatory*. The *pulse* is soft and compressible, from fatty degeneration of the heart, unless the arteries are diseased, which they often are.

4. *Respiratory*. Chronic cough ("drunkard's cough") and more or less dyspnoea on exertion from the bronchial trouble and the weak action of the heart.

5. *Renal*. The evidences of sclerosis of the kidneys are common in old, standing cases; the kidneys are "inadequate" and nitrogenous waste is retained in the blood in consequence.

Diagnosis.—Profound intoxication is sometimes mistaken for—

1. *Apoplexy*. In the latter *one side* is limp and more useless than the other and there is usually no smell of alcohol about the patient (it should be remembered, however, that apoplexy is a result of chronic alcoholism and a vessel may rupture during intoxication.)

2. *Uræmic coma*. In these cases there is albumin in the urine.

3. *Opium poisoning*. The pupils are far more contracted in opium poisoning, the respirations usually slower and there is no smell of alcohol.

Prognosis.—The *prognosis* of *delirium tremens* is usually favorable, but must always be guarded; if it comes on in connection with pneumonia it is usually fatal.

The *prognosis* of chronic alcoholism depends upon (1) the abstemiousness of the patient; (2) the extent of the morbid changes.

Sequelæ.—1. *Digestive.* Cirrhosis of the liver and chronic inflammation of the stomach.

2. *Renal.* Bright's disease, especially the cirrhotic form.

3. *Circulatory.* Fatty degeneration of the heart and sclerosis of the arteries may occur from the changes in the arteries.

4. *Nervous.* Progressive loss of mental power from the increase of connective tissue in the cerebral convolutions; multiple neuritis from the action of the alcohol on the nerves (?); and apoplexy from the changes in the walls of the vessels.

Treatment of *delirium tremens*. The indications are—

1. To *sustain strength*, which may best be done by nutritious and concentrated food and by the use of digitalis when the heart's action is weak; alcohol should be avoided if possible, but it is sometimes necessary.

2. To *allay the craving for alcohol*. Tincture of capsicum and nuxvomica are very useful for this purpose; strong beef or chicken tea, made hot with pepper, is also useful.

3. To *induce sleep*. Paraldehyde, when the stomach will bear it, is best for this purpose. One of the bromides may be given with it with advantage. Opium and chloral are not so safe.

4. To *allay excitement* quiet is necessary. In the way of drugs, paraldehyde, sulphonal, opium, the bromides and chloral have been used. Depressing remedies should be avoided.

CHAPTER IX.

DISEASES OF THE SKIN.

ERYTHEMA.

Definition.—A hyperæmia or superficial inflammation of the skin characterized by redness, with little or no thickening, as a rule, and by the total absence in all cases of vesicles, pustules and scales.

Varieties.—1. Erythema simplex.

2. Erythema multiforme.

3. Erythema intertrigo.

ERYTHEMA SIMPLEX.

Definition.—A hyperæmia of the skin or very superficial inflammation in which there is no exudate and little or no thickening of the skin.

Causes.—1. *Mechanical irritation* from rubbing, pressure, &c.

2. *Heat*, as from exposure of parts which are usually covered to the sun's rays.

3. Certain *chemical irritants*, such as chloroform, mustard, some of the aniline dyes, &c.

4. *Digestive disturbances*, such as the irritation of teething in children (Hyde) and the effects of certain forms of food in some persons.

5. Many drugs, such as antipyrine, belladonna, &c.

Symptoms.—*Appearance.* The skin of the affected part is bright red; there is no swelling and no exudate.

There is sometimes considerable burning and itching.

Diagnosis.—The *diagnosis* is based on the superficial character of the affection and the absence of swelling and exudate.

Prognosis.—The *prognosis* is uniformly good and the trouble is of very short duration.

Treatment.—Treatment is directed chiefly to the *relief of the itching and burning*.

Dusting powders composed of starch and oxide of zinc or subnitrate of bismuth are useful.

Sponging with alcohol and water or camphor and water, or with a solution of bicarbonate of soda, often relieves the itching.

In many cases of "sun burn" the application of cream gives prompt relief. Parts subjected to pressure should be protected by adhesive plaster.

ERYTHEMA INTERTRIGO.

Definition.—A superficial inflammation of the skin where two surfaces lie in close contact.

Causes.—1. *Heat.* It is far more common in hot weather than in cold.

2. *Friction.* It occurs especially if the two surfaces of the skin rub against each other.

3. *Moisture.* The accumulation of sweat between the surfaces aggravates, if it does not cause, the trouble.

4. *Irritating discharges* (urine, and fæces in the case of children, &c.) greatly aggravate the inflammation.

Symptoms.—1. The *usual seats* are the inner surfaces of the thighs, the fold between the buttocks, the under surface of the breasts and the adjacent skin in women, &c.

2. *Appearance.* There is bright redness, with quite sharply-defined borders, and at the bottom of the fold there is often a little crack or fissure in the skin.

3. *Secretory.* There is considerable moisture, which is due, in part to retained sweat and in part to an exudate, which is said not to stiffen the clothing. The secretion is often quite offensive.

4. *Sensory.* There is usually considerable burning and itching.

Diagnosis.—The *diagnosis* is based on (1) the seat; (2) the appearance; (3) the absence of much swelling.

Prognosis.—The *prognosis* is favorable, but recurrences are, of course, frequent, unless the cause can be removed.

Treatment.—1. To *remove the cause* by separating the affected surfaces by absorbent cotton or borated cotton, or by lint smeared with oxide of zinc ointment.

2. To *remove secretion* by gentle but thorough washing with tepid water and soap.

3. To *allay irritation* by dusting powders, of which oxide of zinc and bismuth or starch is one of the best; lycopodium may often be used with advantage. Ointments and lotions are rarely advisable, but the former may occasionally be employed and sometimes the latter. *Cleanliness*, in the case of children, is very important.

ERYTHEMA MULTIFORME.

Definition.—An affection of the skin erythematous in character and characterised by great variety in the forms which it may present.

Varieties.—The principal varieties are (1) annular; (2) marginal; (3) papular; (4) bullous; (5) nodular.

The only one of these requiring special notice is erythema nodosum.

ERYTHEMA NODOSUM.

Definition.—A form of erythema characterized by swellings of considerable size, often an inch or more in width and two or three inches long, elevated above the surface, red in color, hard at first, subsequently becoming soft, but never suppurating.

Causes.—But little is known of any form of erythema multiforme. Erythema nodosum is most common in *early life* and in Spring and autumn. It is probably due to nervous disturbance of some kind.

Symptoms.—1. The *usual seats* are on the front of the tibiae and on the ulnæ, but it may occur on the calves of the legs and elsewhere. It is often symmetrical.

2. *Size and color.* These features have already been described.

3. *Sensory.* Pain and tenderness are always present in a greater or less degree, and are due probably to pressure on the nerves.

4. *General.* A general malaise, slight fever and sore throat are common accompaniments.

Duration.—The enlargement usually lasts about ten days and then disappears.

Diagnosis.—The *diagnosis* is based on the absence of any history of injury, the symmetrical character of the lesion and the course of the disease.

Prognosis.—The *prognosis* is good.

Treatment.—1. *Local.* Application of lead lotion is advised. Alcoholic lotions are also useful in many cases.

2. *Constitutional.* Iron and strychnine have been used with advantage, and also the mineral acids. Disturbances of digestion, if present, should be corrected.

URTICARIA.

(Nettle rash. Hives.)

Definition.—An inflammatory, non-contagious affection of the skin characterized by the sudden formation of wheals, accompanied by intense itching.

Causes.—The *essential cause* is probably some disturbance of the vaso-motor nerves.

The *exciting* causes are—

1. *Irritants*, such as the bites or stings of insects.
2. *Certain drugs*, such as quinine or copaiba, which will cause an attack in some persons.
3. Certain articles of *diet*, especially shell fish; but some persons are affected by other articles, such as strawberries, &c.
4. *Exposure to cold* will often bring on an attack (Hyde).
5. It may be caused *reflexly* from disease of other organs, as the kidneys, uterus, &c.

It is sometimes associated with or alternates with attacks of asthma.

Pathology and Morbid Anatomy.—The disease is almost certainly due to vaso-motor disturbance. The wheals contain a sero-fibrinous exudate which compresses the vessels and forces out the blood.

Symptoms.—1. The *onset* is sudden, the wheals suddenly appearing and as suddenly disappearing.

2. The *wheals* vary from the size of a finger-nail to the diameter of an inch or, rarely, more. They may appear on any part of the body except the face, scalp and soles of the feet. In *color* they are usually white with a reddish border. In number, there may be but one or two or the body may be covered with them.

3. *Sensory. Itching* is intense, probably in consequence of the irritation of the peripheral nerves.

Diagnosis.—The *diagnosis* is based on the appearance and itching, and on the sudden occurrence and equally sudden disappearance of the wheals.

Prognosis.—The *prognosis* is good in acute cases; in chronic cases it is very rebellious to treatment.

Treatment.—1. To *remove the cause* by (1) correcting digestive disturbances with calomel, alkalies, the mineral acids, nux vomica; (2) preventing irritation of the skin by rough flannels.

2. To *relieve itching* by (1) bromides, antipyrine, &c., *internally*, and (2) by chloral solutions, camphor, starch, bismuth, fluid extract of grindelia robusta, lime water, &c., *externally*.

ECZEMA.

Definition.—An inflammatory affection of the skin characterized by redness and the formation of papules, vesicles, pustules or scales. As a rule, these lesions are more or less combined in each case.

Forms.—The disease may be (1) acute, or (2) chronic.

Causes.—The causes are exceedingly obscure. Some persons are *predisposed* to eczema, but it is not known in what this predisposition consists.

Irritants, such as rough clothing, exposure, scratching, and various other things may occasion it.

Certain plants, as *poison oak* and *poison ivy*, will induce it.

Dyspepsia will sometimes bring on an attack, and *gouty* persons seem to be especially liable to it. *Asthma* is a common complication.

Morbid Anatomy.—Eczema is an inflammatory condition, and there is redness, heat and swelling, as in all other inflammations.

The exudate may be *fibrinous*, as in the *papular* variety, *serous* in the *vesicular*, *purulent* in the *pustular*; in the squamous form, which is very chronic usually, the amount of exudate is too slight to be perceptible, but sometimes the skin is thickened and there is often atrophy of the sweat glands and hair follicles.

Symptoms.—1. The *appearance* depends upon the form (papular, vesicular, pustular or squamous). There is redness of the skin always.

In the *papular* form, small papules appear on a reddened base.

In the *vesicular*, little vesicles filled with serous fluid are seen.

In the *pustular* form there are little pustules.

In the *squamous* form the skin is thickened and scaly.

2. The *seat and extent* of eczema varies greatly in different cases; it may occur on any part of the body; the head and face is a favorite seat in the case of children; the hands, genital organs and anal region are often affected later in life. The *extent* varies also very greatly; the patch may be not more than an inch in diameter or a large part of the body may be affected.

3. *Secretory*. In all, or nearly all, cases at some time in their course there is more or less secretion; in acute cases it is usually abundant, in chronic cases, scant. If the secretion (or exudate) is serous it dries into scales, if purulent, into thick and friable scabs.

4. *Sensory*. *Itching* is a prominent symptom in all cases, and sometimes is so violent as to cause intense annoyance.

Diagnosis.—Eczema is distinguished from (1) *acne* by the more superficial seat of the inflammation and the greater prominence of the itching in the former; (2) *impetigo* by the absence of itching, as a rule, in this disease, and the larger size and more scattered ar-

rangement of the pustules; (3) *scabies* by the presence of the burrows and parasites peculiar to this disease.

Prognosis.—The *prognosis*, as to life, is always good; the *duration* exceedingly uncertain and *relapses* frequent.

Treatment.—The *treatment* depends upon the *stage* of the disease, but even in cases apparently similar in all respects remedies which are efficacious in one will fail in another.

The first indication, which holds good in all cases, acute and chronic, is—

1. To *remove the causes* of the disease and *all sources of irritation*, such as (1) local irritants, coarse flannel or friction from any cause; (2) frequent washing of the inflamed spots, especially with strong soaps; (3) disturbances of the digestive organs. Bismuth and the alkalies are useful in many cases.

2. To *remove crusts*, when present, by poultices or oily applications.

3. To *relieve inflammation* and *allay itching*—

A. In *acute* cases by *sedative* applications, such as (1) Carron liniment; (2) ointment of subacetate of lead; (3) menthol dissolved in sweet oil; (4) fluid extract of *grindelia robusta*; (5) a paint made of bismuth and glycerine; (6) oxide of zinc ointment, sometimes diluted; (7) carbolic acid and sweet oil (1 or 2 grains to the ounce); (8) powdered starch; (9) powdered boracic acid alone, or combined with bismuth or starch; (10) oxide of zinc and starch or bismuth.

B. In *chronic* cases, by more or less *stimulating* applications, such as (1) tar ointment; (2) oil of cade diluted with olive oil; (3) green soap; (4) ichthyol ointment; (5) sulphur ointment, &c.

Arsenic should never be used internally (or externally) in the acute forms of eczema; in the chronic forms it is often very useful.

IMPETIGO.

Definition.—An inflammatory affection of the skin characterized by the formation of discrete pustules, the size of a coffee bean or larger.

The disease is non-contagious and is by many considered to be a form of eczema.

Causes.—*Irritation*, by scratching with dirty finger nails, is probably a cause.

2. *Age.* The disease is most common in children and young adults.

3. *Season* of the year. It occurs generally in the autumn.

Morbid Anatomy.—1. *Size and shape.* The pustules are the size of a coffee bean or larger, rounded or oval in shape and elevated above the surface of the skin.

2. The *exudate* is purulent.

Symptoms.—1. The characters of the pustules have just been stated.

2. The numbers vary exceedingly, usually there are only eight or ten.

3. The *usual seats* are on the face—especially about the mouth—and on the feet.

4. The *surrounding skin* is not inflamed, as a rule, but in *ecthy-ma*, which is now often considered a form of *impetigo*, there is a red areola around the pustules.

5. *Sensory.* *Itching* is not usually prominent.

6. *Terminations.* The pustules dry up and form scabs or crust; sometimes they rupture.

Diagnosis.—From ordinary *eczema*, by the absence of great itching and of a vesicular stage and by the scattered arrangement of the pustules: (sometimes, however, especially around the mouth, they merge into each other).

Prognosis and Duration.—The *prognosis* is good; the *duration* rarely more than a few weeks.

Treatment.—The *treatment* consists in—

1. The *evacuation of the pus*.

2. The use of *antiseptic lotions* or *ointments* containing corrosive, sublimate, carbolic acid, resorcine, boracic acid or some agent of this class.

LICHEN PLANUS.

Definition.—An inflammatory, non-contagious affection of the skin characterized by the formation of raised, flat patches of a deep purple color, glistening in appearance and but slightly desquamative.

Causes.—1. Probably it is intimately connected with some *ner-vous disturbance*, as it is often associated with neuralgia.

2. *Age.* It is most common in early and middle adult life.

3. It is *not contagious*.

Morbid Anatomy.—1. The *hair follicles* are primarily involved, the inflammation commencing in the outer layer of the root sheath (Hyde).

2. *Papules* are formed first and the flat-raised spots are formed by coalescence of the papules.

Symptoms.—1. *Characteristics* of the rash. Flat papules, from the size of a pin's head to that of a split pea, deep purple color, glazed, umbilicated, with but little tendency to desquamation and pursuing a very chronic course and leaving a pigmented spot.

2. *Seat and extent.* They may occur at any part of the body, are most common about the flexor surfaces of the wrists and knees and may be widely distributed.

3. *Sensory.* The *itching* is usually moderate, but is sometimes quite severe.

Diagnosis.—The *diagnosis* is based on (1) the polygonal shape; (2) the purple color; (3) the umbilication.

Course, Duration and Prognosis.—The course is chronic, the duration often many years and the prognosis, as to life, good, but as to permanent recovery, not very favorable.

Treatment.—A. *General.* 1. Arsenic is very useful in most cases, especially the very chronic ones.

2. *Tonics.* Cod liver oil, iron, zinc, &c., are useful.

B. *Local.* Ointments of (1) corrosive sublimate; (2) carbolic acid; (3) tar; (4) ichthyol; (5) thymol; (6) chrysophanic acid.

LICHEN RUBER.

The disease is, in appearance, much like the one just described, except that the eruption has a "marked tendency to generalization and the induction of a fatal marasmus" (Hyde).

The *causes* of the disease are unknown.

The *symptoms* in the beginning are like those of lichen planus, but later on the skin becomes greatly thickened and cracks and is covered with scales.

The *diagnosis* is based upon the symptoms named.

The *prognosis* is always serious.

The *treatment* consists in the administration of *arsenic*, tonics and good diet, and the local use of remedies to allay irritation.

PRURIGO.

Definition and Frequency.—A very rare affection of the skin, chronic in character and characterized by the occurrence "on the exterior surfaces of the limbs and on the trunk of minute, pale or reddish papules with extensive infiltration and intolerable itching."

Causes.—1. *Prevalence.* The disease is very rare and is confined chiefly to Austria (Hyde).

2. *Bad hygienic conditions* are undoubted causes.

3. *Age.* It begins, usually, in infancy, and persists through life.

4. It is *not* contagious.

Symptoms.—1. *Characteristics of the rash.* Papular, pale or reddish in color, with extensive infiltration.

2. *Seat and extent.* The external surfaces of the legs and thighs or of the arms are affected first; the trunk later. The glands are frequently enlarged.

3. *Sensory.* Itching is extremely severe, and interferes with sleep and rest so much as to lead occasionally to insanity.

Diagnosis.—1. From *eczema* by the seat, uniformity of the rash and the obstinate course.

2. From *scabies* by the absence of burrows and the seat.

Prognosis.—The *prognosis*, as to recovery, is bad; it usually lasts through life.

Treatment.—1. *General.* To improve the general health as far as possible by fresh air, good food and tonics. Arsenic is not advisable.

2. *Local.* Sulphur, tar and green soap have been used with some relief.

 PITYRIASIS RUBRA.

(Exfoliative Dermatitis.)

Definition and Frequency.—A very rare, chronic, inflammatory affection of the skin characterized by redness and profuse scaling and by more or less itching.

Causes.—The *causes* are unknown.

Morbid Anatomy.—The chronic inflammation leads to increase of connective tissue and atrophy of the glandular structures and hair follicles.

Symptoms.—*Characteristics of the rash.* Bright red in color and marked by the exfoliation of *innumerable scales*. No exudate unless the skin cracks at the joints, as it sometimes does.

2. *Sensory.* Itching varies in degree; it is often severe.

Diagnosis.—1. From *eczema* by the absence of exudation, vesicles or papules at any time.

2. From *psoriasis* by the diffuse character of the rash.

Prognosis and Duration.—The *prognosis* is serious, as to life, in bad cases, the disease often leading to nephritis, and the *duration* is usually life long.

Treatment.—No internal medication has done any good, as a rule; in a single case, carbolic acid, one drop at a dose, was apparently beneficial.

Locally, vaseline is comforting.

PSORIASIS.

Definition and Frequency.—A chronic, non-contagious, inflammatory affection of the skin characterized by the formation of flat papules covered with white scales.

It is one of the most frequent of the skin diseases.

Causes.—The *causes* are unknown; it occurs at all ages, after infancy, and in both sexes.

Symptoms and Signs.—1. *Characteristics of the rash.* Flat papules from $\frac{1}{8}$ th to $\frac{1}{2}$ inch in size, red in color, but covered with white scales.

2. *Seats and extent.* The most common and earliest seats are on the posterior surface of the elbows and the anterior surface of the knees; it is seen also on the limbs, back and face.

3. *Sensory.* Itching is not usually marked.

Constitutional symptoms are absent.

Diagnosis.—1. From *eczema* by the absence of moisture at any period and the absence of itching to any marked degree.

2. From *pityriasis*—(See Pityriasis).

Prognosis.—The *prognosis*, as to life, is *always good*, but the *duration* is uncertain and *relapses* are certain.

Treatment.—A. *General.* Arsenic is most useful. Cod liver oil and iron in case of debility.

B. *Local.* Tar ointment, pyrogallie acid ointment, (1 drachm to 1 ounce) chrysophanic acid ointment (5 to 10 grains to 1 ounce), hot baths and soap.

PEMPHIGUS.

Definition and Frequency.—A rather rare, acute or chronic affection of the skin, characterized by the formation of bullæ from the size of a bean to that of an egg.

Causes.—The *causes* are practically unknown. The disease is *not* contagious and is most frequent in children.

Syphilis will cause a bullous eruption, which is not usually classed with pemphigus.

Symptoms.—1. *Characteristics of the eruption.* Papules soon succeeded by bullæ varying in size and containing serous, rarely sero-purulent or bloody, fluid.

2. *Seats and number.* The bullæ may occur at any part of the surface, and there may be only a few bullæ or a hundred or more.

3. *Sensory.* Itching is slight in most cases, but is rarely very troublesome.

4. *Constitutional symptoms*—fever and sometimes prostration.

5. *Termination.* The usual termination is in drying and crusting, but the bullæ may burst and form sores.

Diagnosis based on the presence of bullæ. In infants newly born, the disease is nearly always syphilitic. (Pye-Smith).

Prognosis.—The *prognosis* is serious in infants and old persons; favorable in others.

Treatment.—A. *General.* Arsenic is most useful; tonics and iodide of potassium, if necessary.

B. *Local.* Of little value; oxide of zinc ointment has been advised.

HERPES ZOSTER.

(Shingles. Zona).

Definition.—An affection of the nerves, nerve centers or ganglia, characterized by the formation on the skin of firm, distended vesicles, limited to an area supplied by one or more twigs of a cranial or spinal nerve.

Causes.—1. The *essential cause* is an inflammation of the nerve, sensory nerve root or ganglion.

2. *Age and sex* have but little influence.

3. *Cold and dampness* seem to be causes occasionally.

4. *Injuries* involving a nerve may cause an attack.

Symptoms.—1. *Characteristics of the eruption.* Maculæ first appear, which are soon followed by vesicles as large as a pea or smaller, containing serum usually, but sometimes pus or blood.

2. *Seats and extent.* The body (intercostal nerves), face, head, eyes, arms, genital organs, &c., may be involved. The eruption is limited to the area of distribution of one or two nerve twigs.

3. *Nervous.* Neuralgia is nearly always present; it may precede the eruption and be very intense; tenderness is also present; there is little or no itching, but may be numbness and various paræsthesiæ.

4. *Constitutional symptoms*—fever, anorexia, &c.—are rare, but may occur.

5. *Terminations.* The vesicles *rarely burst*, but usually dry up and form a scab which finally comes off and leaves a scar.

Diagnosis.—From *eczema* by the thickness of the wall of the vesicle, the absence of itching and the presence of neuralgia.

Prognosis usually favorable, but occasionally the disease persists and the patient is worn out by suffering. The usual *duration* is two or three weeks.

Treatment.—A. *General.* Phosphide of zinc and extract of nux vomica, quinine, strychnine and arsenic have been used.

B. *Local.* Oleate of morphia, oleate of zinc, menthol dissolved in olive oil and various dusting powders containing oxide of zinc, &c., have been used; the object is to relieve pain.

Galvanism sometimes does good. The skin should be protected from rubbing.

Herpetic eruptions are of common occurrence on the lips (in fevers) and on the genital organs; they are of little moment.

ICTHYOSIS.

Definition.—A deformity of the skin appearing in the first years of life (and probably congenital) characterized by the formation of scales or plates of horny consistency.

Causes.—I. *Age.* It is almost certainly congenital, but appears usually in the first year of life.

2. *Heredity* appears to be a cause, but opinions differ on this point.

Symptoms and Signs.—1. *Appearance.* The skin may be simply very dry, with slight scaliness, or the scales may be thick and in polygonal masses. In still more severe cases the skin may be horny from the development of the papillæ.

2. *Seats and extent.* The most common seat is on the extensor surfaces of the limbs and on the back, but nearly the whole body may be affected—the face and genital organs least.

3. *Secretory.* There is little or no secretion of sweat.

4. *Sensory* symptoms are not usually marked.

5. *Constitutional* symptoms are also slight or entirely absent; occasionally growth seems to be partially arrested by it.

Diagnosis.—The *diagnosis* is based on the symptoms and the absence of inflammation.

Prognosis.—The *prognosis*, as to life, is good; as to recovery, bad.

Treatment.—A. *General.* The affection is a deformity, and internal treatment is unavailing. A *warm climate* to stimulate the function of the skin is advisable.

B. *Local.* Baths, alkaline or simple, are useful in order to soften and remove the scales; after bathing, ointments, either simple vaseline or some bland ointment, are useful, and also the glycerole of starch.

SCLERODERMA AND MORPHŒA.

Definition and Frequency.—*Scleroderma* is a more or less chronic disease, characterized by hardening of the skin over extensive areas.

Morphœa is an affection of the skin, characterized by a gradual hardening in patches. Both diseases are *extremely rare*.

Causes.—1. Both diseases (they are probably different forms of the same disease) are *tropho-neuroses*.

2. *Age.* The affections are most common in young adults, but may occur at any age.

3. *Sex.* Women are far more liable to this disease than man.

Morbid Anatomy.—In *scleroderma* there is a gradual *increase of connective tissue* in the corium and sub-cutaneous tissue.

In *morphœa* there is a similar change, less generalized.

Symptoms.—1. *Appearance.* The skin in *scleroderma* becomes whitish in color; in *morphœa* there may be redness or yellowness at first, and sometimes pigmentation.

2. *Seat and extent.* *Scleroderma* may involve large surfaces, the face and neck are usually involved first. *Morphœa* occurs in patches, often on the distribution of a nerve.

3. *Nervous.* Various *paræsthesiæ* may be present in cases of *morphœa*. Stiffness is troublesome in bad cases of *scleroderma*, from increase of connective tissue.

4. *Temperature.* The temperature over the affected spots is depressed, because of the anæmic condition.

5. *Termination.* Often recovery occurs most unexpectedly. Death may occur from marasmus, or from stiffening of the jaws (Fagge.)

Diagnosis.—The *diagnosis* is simple.

Prognosis.—The *prognosis* is uncertain and the *duration* variable.

Treatment.—A. *General.* Nerve tonics such as cod liver oil, iron, quinine and strychnine are advised. Arsenic has been found useful.

B. *Local.* The *galvanic* current has given good results in a number of cases. Massage, baths and mercurial ointment are recommended (Duhring).

LUPUS VULGARIS.

Definition.—A very chronic tubercular affection of the skin, characterized by redness and the formation of nodules and ulcers.

Causes.—1. The *bacillus tuberculosis* (see Tuberculosis) is the essential cause.

2. *Age.* It commences as a rule in childhood.

3. *Sex.* Females are affected oftener than males.

Morbid Anatomy.—1. The *blood vessels* are engorged, causing redness, and tubercles are formed underneath the epidermis. *Ulceration* occurs at a later stage.

2. The *exudate* is purulent and rather scant, forming yellowish crusts or scabs on the ulcers.

3. The *tubercles*, unlike those in ordinary tuberculosis elsewhere, are rich in blood vessels (Green).

Symptoms.—1. *Appearance.* *Redness*, more or less bright and ending quite abruptly; firm *elevations*, later on *ulcers* covered with crusts.

2. *Seat and extent.* The face is usually involved, and especially the nose, but it may occur elsewhere. The size of the patches varies from an inch to five or six inches in diameter.

3. *Sensory.* Sensory symptoms are absent, as a rule.

4. There are *no constitutional* symptoms, as a rule, either, unless complications exist.

5. *Termination.* *Cicatrization* often occurs at one point while it is spreading at another; the cicatrices are usually soft and superficial.

Diagnosis.—The *diagnosis* is usually easily made; in doubtful cases Koch's lymph has been used to decide the matter.

Prognosis and Duration.—The *prognosis* as to life is good, as to recovery bad; the *duration* usually being many years.

Treatment.—A. *General.* Tonics, such as cod-liver oil, iron and hypophosphites are advised.

The *injection of Koch's lymph (tuberculin)* has recently been employed with apparently curative results, but its value is not positively determined.

B. *Local.* Incisions, punctures and the galvano-cautery have been used to destroy the blood vessels and cause an atrophy (?) of the tuberculosis tissue. Scraping out the ulcers is also practised with benefit.

Ointments of sulphur, mercury, pyrogallol and other substances have been employed.

LUPUS ERYTHEMATODES.

Lupus erythematodes is probably a more superficial form of *lupus vulgaris*, in which nodules form, but no ulcers. It occurs usually in *middle life*, commonly in women, and the usual seat is on the *nose* and *cheeks*, but it may be extensively distributed. It is characterized by *redness* and the formation of *thin scars* without ulceration. It lasts for years. The *treatment* is like that of *lupus vulgaris*.

LEPROSY.

(Lepra. Elephantiasis Græcorum.)

Definition.—An infectious disease, very chronic in course, and characterized by the formation of nodules on the skin, larynx, &c., or by neuritis, with anæsthesia, and later by ulceration.

Varieties.—1. Tubercular. 2. Anæsthetic.

Causes.—1. A *germ*. The *bacillus lepræ*, which is precisely like the bacillus tuberculosis (q. v.) except that it stains more readily (Crookshank) and inoculation with it does not cause tuberculosis.

2. The *mode of conveyance* is probably in the secretions.

3. The *avenue of introduction* is not known.

4. *Males*, in middle life, are rather more subject to the disease than others.

5. *Distribution*. The disease occurs in the Sandwich Islands, China, Japan, India and a number of other countries; it is rare in America and England.

Morbid Anatomy.—1. In the *tubercular* form elevations, nodules are formed the size of a bean or very much larger, consisting chiefly of granulation tissue, with some blood vessels; these nodules occur in the skin and mucous membranes, notably in the larynx, and they may *ulcerate* and sometimes *cicatrize*.

2. In the *anæsthetic* form there is a *neuritis*, involving one or more nerves and leading to atrophic changes in the skin and muscles.

Symptoms.—A. *Prodromic*. Attacks of fever and digestive disturbances, depression and anorexia of variable duration and at variable intervals.

B. Of the *developed* disease—

1. *Appearance*. There may be (1) nodules (or tubercles), varying in size from that of a bean to a tomato—yellowish or reddish in color, or (2) maculæ, of reddish or white color, or (3) large surfaces of skin looking white and atrophied.

Later, contraction of the eyelids, cheeks, lips and other parts, and ulceration leading to loss of fingers or toes or the entire hand may occur.

2. *Seat and extent*. The most usual seat of leprosy is on the face or hands, but other parts of the body may be affected.

3. *Sensory*. Various paræsthesiæ occur and later complete anæsthesia.

4. *Constitutional*. There is a gradual loss of strength.

Diagnosis.—The *diagnosis* is based on the character of the lesions and their great chronicity, and on the bacillus lepræ.

Prognosis.—The *prognosis* is uniformly bad; the *duration* in the tubercular form from eight to ten years, in the *anæsthetic* form from fifteen to twenty years.

Treatment.—A. *Prophylactic*. Isolation, to prevent a further spread.

B. *Remedial*. Change of residence to a country where the disease does not prevail; tonics are suggested, but have little effect. Baths and antiseptic ointments are often of service.

ACNE.

Definition.—A chronic, inflammatory affection of the sebaceous glands and peri-glandular tissue, characterized by the formation of small papules or pustules. It is probably the most common of all skin diseases.

Causes.—1. *Age and sex*. It usually occurs about the age of puberty and is rather more common in boys than in girls.

2. Possibly *irritation* of the skin may cause it.

3. *Certain drugs*—the bromides and iodides—will bring on acne, when taken for any length of time.

Morbid Anatomy.—The sebaceous glands and the peri-glandular tissue is inflamed and then results redness and swelling.

The *exudate* may be sero-fibrinous or *purulent*, usually it is purulent.

Symptoms.—1. *Appearance*. The eruption is red in color, but often becomes yellow (when pus forms) and frequently a black spot is seen in the centre of the bump, in consequence of the stopping of the mouth of the follicle with foreign matter. The bumps vary in size from $\frac{1}{16}$ inch to $\frac{1}{8}$ inch in diameter.

2. *Seat and extent*. The usual seats are on the face and between the shoulders. There may be only a few bumps or the face may be covered with them.

3. *Sensory* symptoms are absent, as a rule, but there may be some sensitiveness.

4. *Constitutional* symptoms are often absent, but there may be anæmia and various digestive disturbances.

5. *Terminations*. As a general rule, no scars are left, but in severe cases there may be extensive pitting.

Diagnosis.—The *diagnosis* is based on the character of the eruption and the absence of sensory symptoms.

Prognosis and Duration.—The *prognosis* is good; the *duration* often several months.

Treatment.—A. *General.* To correct any disturbance of the general health by purgatives, if constipation exists, and by cod liver oil, iron and other tonics if there is anæmia or debility.

B. *Local.* Pus should be let out by small punctures and pressure; the skin should be thoroughly rubbed with a towel wrung out of hot water and solutions of bichloride of mercury (1 grain to the ounce), sulphide of potassium (1 drachm to 4 ounces) or ointments of sulphur ($\frac{1}{2}$ drachm to 1 ounce of vaseline) or resorcine (10 to 20 grains to the ounce of vaseline) should be applied.

ACNE ROSACEA.

Definition.—A chronic affection of the skin occurring chiefly on the face—especially on the nose—and characterized by passive hyperæmia and subsequently by more or less enlargement from increase of connective tissue and inflammation of the sebaceous follicles.

Causes.—1. Uterine or ovarian disturbance in women, especially at puberty and the menopause.

2. *Alcoholic liquors.*

3. Exposure to *heat* or *cold*.

Symptoms.—1. *Appearance.* First there is simply redness of the affected part; later, large vessels may be seen in the skin and still later there may be great hypertrophy of the part with the formation of nodular masses. Ulceration and scab formation are never observed.

2. *Seat.* The most common seat is the nose, but the cheeks and the forehead may also be involved.

3. *Sensory* symptoms are absent; *constitutional* symptoms of various kinds may occur from the causative condition.

Diagnosis.—It is distinguished from other skin diseases (syphilis and lupus) by the absence of crusts or ulcers.

Prognosis and Duration.—The *prognosis* of the mild form or in the early stage is good; when nodular masses have formed there is no possibility of their removal except by surgical means. The *duration* depends on the form.

Treatment.—A. *General.* The chief object of general treat-

ment is to lessen the amount of blood in the face. Alcoholic liquors should be forbidden, a scant diet advised, disturbances of the uterine functions corrected and purgatives should be administered if there is constipation.

B. *Local.* To *cause contraction of the dilated vessels* and stimulate the skin by sulphur ointment or solution, green soap, alcoholic solution of corrosive sublimate (1 grain to the ounce), &c.; or to *destroy the enlarged vessels* by galvano puncture or puncture with a small knife.

SYCOSIS.

Non-parasitic sycosis is a rare disease characterized by an inflammation of the hair follicles and the formation of papules or pustules.

The *causes* are unknown.

It occurs on the face—on the chin, cheek or upper lip—and causes considerable burning pain. A hair rises from the center of each papule or pustule.

It is distinguished from *eczema* by its seat and the absence of itching, and from *parasitic sycosis* by the absence of a parasite.

The disease persists for a long time if not properly treated.

The *treatment* consists in the administration of cod liver oil, iron and arsenic, shaving and the application of oxide of zinc ointment in the acute cases or of sulphur ointment at a later period.

TINEA CIRCINATA.

(Ring worm.)

Definition.—A parasitic affection of the skin, contagious and characterized by more or less circular, squamous, vesicular, papular or (rarely) pustular spots.

Causes.—1. The *essential cause* is a parasitic plant, the *tinea trycophyton*, composed of cells secured end to end in such a way as to form long and branching plants. It forms *spores* from which new plants develop in about twelve days.

2. *Heat and moisture* are favorable to the growth and development of the plant.

3. The disease may be *transmitted from animals*.

4. *Age.* It is more common in children than in adults, but may occur at any age.

5. It is highly *contagious*.

Morbid Anatomy.—The *trycophyton* grows between the superficial layers of the epidermis.

Symptoms.—1. *Seat and extent.* It may occur at any part of the body, but is especially common on the face and thighs.

When the *scalp* is attacked it is called *tinea tonsurans*; when it involves the follicles of the beard it is known as *parasitic sycosis*.

A *patch* is rarely more than 3 or 4 inches in diameter and is usually much less.

2. *Appearance.* Patches of *tinea circinata* are circular in outline, reddish in color, papular, vesicular, rarely pustular, but most frequently scaly—the scales being small and branny.

In *tinea tonsurans* the hair is lost and *baldness* in patches results.

3. *Sensory.* As a rule, there is little itching, but it may be intense.

Eczema marginatum is a form of ring worm occurring usually on the buttocks and thighs, but sometimes on other parts of the body, and characterized by a red, sharply-defined and usually elevated ring, which gradually extends and is attended with violent itching.

Diagnosis.—It is distinguished from *psoriasis*, *eczema* and all other eruptions by a microscopic examination, when the *trichophyton* can readily be seen.

Prognosis.—The *prognosis* is good, but *eczema marginatum* is often tedious and obstinate.

Treatment.—1. To *remove all scales* by thorough bathing and rubbing with soap and water.

2. To *destroy the parasites* by applying tincture of iodine, bichloride of mercury solution (1 grain to the ounce), thymol or boracic acid solutions and ointment of nitrate of mercury (30 grains to the ounce) chrysarobin or pyrogallol (5 to 10 grains to the ounce of vaseline).

SCABIES.

(Itch.)

Definition.—An inflammatory and highly contagious affection of the skin caused by the itch parasite (*acarus scabiei*).

Causes.—1. The *essential cause* is an animal parasite—the *acarus scabiei*—about $\frac{1}{10}$ th of an inch in length and oval in shape.

The females only, penetrate the skin, the male, which is much more short-lived, remaining on the surface.

2. *Want of cleanliness* is a frequent, but not an invariable, cause of scabies.

3. *Age and sex.* Children are more liable to the disease than adults and men more than women, because they are brought into more intimate contact with each other.

The disease is very *contagious*—the parasites readily passing from one body to another.

Symptoms —1. *Seat and extent.* All parts of the body may be attacked, the fingers and clefts between the fingers, the toes, the axillæ, the breasts in women and the penis in men are most liable to be attacked—the face least liable.

2. *Appearance.* Papules, vesicles or occasionally pustules may be formed, but the characteristic lesion is a *burrow* in the skin made by the parasite, and containing eggs—a dozen or more—black spots (fæcal matter) and at one end a white spot—the itch mite—which may be removed with a needle and examined under the microscope.

3. *Sensory.* Itching is usually excessively severe, especially at night, because the warmth of the bed renders the nerves more irritable.

Diagnosis.—The *diagnosis* is based on (1) the burrows; (2) the situation of the eruption; (3) the presence of the parasite.

Prognosis.—The *prognosis* is always good.

Treatment.—1. *Thorough cleansing* of the surface with soap and water to remove all obstacles to the action of parasitocides.

2. The *application* of *parasitocides*, such as an ointment of sulphur, 60 grains, Peruvian balsam, 120 grains, and vaseline, one ounce—applied every night for three days; this treatment is practically certain to effect a cure.

CHAPTER X.

DISEASES OF THE NERVOUS SYSTEM

GENERAL SYMPTOMATOLOGY OF DISEASES OF THE NERVOUS SYSTEM.

Disturbances of Motion.—A. *Motor irritation* may show itself in the form of—1. *Spasm*. 2. *Tremor*. 3. *Ataxia*, or loss of co-ordinating power.

Spasm or convulsions may be (1) *direct*, when the irritation causing them is applied directly to the motor centre or centres, or to the motor nerve, or to the muscles themselves; or (2) *reflex*, when spasm is due to an irritant applied to a distant point, the impression then being conveyed to the nerve centre and reflected to the muscles.

Spasms are said to be *tonic* when they are continuous for a greater or less length of time. They are called *clonic* when they are interrupted or jerking in character.

They are classified, also, according to the *extent* of the spasmodic movements; for example, the spasms may be (1) *general*, as in tetanus and strychnia poisoning; (2) confined to *one side* of the body (hemi-spasm); (3) a *single muscle* may be affected (mono-spasm), or (4) a number of muscles may be affected irregularly, as in chorea, &c., &c.

By *tremor* is meant a tremulous motion of different parts of the body from muscular contractions of very slight excursion. It may be *constant* as in paralysis agitans or occur *only when the person tries to move*, as in insular sclerosis; this latter form is sometimes called “tremor of intention.”

By *ataxia* is meant a loss of the power to move different muscles together and in harmony with each other, as in locomotor ataxia.

B. *Weakness or loss of motor power*. There may be simply a weakness of motor power, which is called *paresis*, or a *complete* loss of power, which is called *paralysis*.

The extent and distribution of paresis or paralysis varies in different cases. For example, *hemi-plegia* is paralysis of one lateral half of the body, *paraplegia* is paralysis of the lower limbs and lower part of the trunk, *monoplegia* is paralysis of a single muscle or group of muscles.

The general causes of paralysis are—

1. Certain lesions of the *motor centres* in the brain, due to hemorrhage, embolism, tumors, &c. Paralysis from this cause is not attended by any sensory disturbance, as a rule, it varies in extent and is frequently followed by *secondary degenerations*, q. v.

2. Certain lesions affecting the *motor tracts* in the brain or cord. Lesions of these tracts, as a rule, cause hemiplegia (not monoplegia) and are frequently attended by disturbance of sensation (hemi-anæsthesia) because the motor tracts occupy the anterior two-thirds of the posterior limb of the internal capsule and the sensory tracts the posterior third and the internal capsule is a common seat of hemorrhage.

3. Disease and loss of function of the large *multipolar cells* in the *anterior cornua* of gray matter.

4. Certain injuries or diseases of the *motor nerves*, such as section of a nerve and neuritis. In these cases there is a degenerative change in the nerve fibres and in the muscles which they supply. Sensation is more or less affected if the nerve be a mixed one, and the paralysis is of limited extent, but several groups of muscles may be involved, as in multiple neuritis.

5. Certain *poisons*, such as lead and arsenic. They probably act by setting up a neuritis.

6. Certain *acute infectious diseases*, such as diphtheria and typhoid fever; they also probably cause neuritis.

7. Certain *reflex* forms of paralysis have been described, but it is very doubtful whether such cases occur.

8. Certain cases of *hysteria*; this is a common cause of paralysis, and it may be limited in extent or quite extensive. Disturbances of sensation often accompany the paralysis, but there are no trophic disturbances.

Disturbances of Sensation.—Sensation may be (1) increased (*hyperæsthesia*); (2) lost (*anæsthesia*) or (3) perverted in various ways (*paræsthesia*). *Hyperæsthesia* may be due to (1) increased irritability of the sensory nerve fibres; (2) increased irritability of the sensory centres.

Anæsthesia may be due to the same general causes which would lead to *motor* paralysis, the *sensory* centres and tracts being involved in cases of anæsthesia instead of the motor. Furthermore, there may be loss of the sense of pain (analgesia) without loss of tactile sense.

Paræsthesia may assume a number of forms, such as tingling, numbness, formication, feelings of constriction, sensations of heat and cold, &c.

Disturbances of the Reflexes.—Both *cutaneous* and *tendon* reflexes may be (1) increased; (2) diminished or lost.

The conditions which *increase* the reflexes are—

1. Cutaneous hyperæsthesia.
2. Excessive irritability of the nerve centers.
3. Increased irritability of the muscles.
4. Paralysis of or separation from the inhibitory centers. (Setchenow's center).

The pathological conditions which cause a *prolonged* or *permanent* diminution or loss of some of the reflexes are—

1. Functional inactivity of sensory nerve terminals.
2. Functional inactivity of sensory nerve fibres.
3. Functional inactivity of reflex centers.
4. Functional inactivity of motor nerves in the cord.
5. Atrophy of multipolar cells in the anterior horns of gray matter.
6. Functional inactivity of motor nerve fibres.
7. Muscular atrophy.

Temporary diminution or loss of reflexes may be due to irritation of a sensory nerve or to shock.

Disturbances of Nutrition (Trophic lesions).—Trophic lesions may result from affections of (1) the brain; (2) the spinal cord, including the medulla oblongata; (3) the nerves.

Secondary Degenerations.—*Lesions of the large cells* in the motor centers of the cerebral cortex or in the fibres leading downwards into the cord from these cells will cause atrophy and degeneration in the motor tracts of the cord below the seat of lesion, but the nerves themselves which pass out from the spinal cord will not be affected, nor will the muscles. There is an increase of connective tissue in the motor tracts of the cord after the atrophy and degeneration of the nerve fibres. These changes in the cord are due to the separation of the motor tracts from the cells in the motor centers, and are called "*Secondary degenerations.*"

Lesions of the spinal cord may cause nutritive disturbances of the muscles, nerves, bones, joints, connective tissue and skin.

Muscular Atrophy.—Trophic disturbances of the *muscles* and probably also of motor nerves are always due to lesions of the large multipolar cells in the anterior cornua or of the fibres leading therefrom (as in acute anterior poliomyelitis and progressive muscular atrophy).

Trophic Affections of the Bones, Joints, Skin, &c.—Nutritive disturbances of *bones and joints* (such as those which occur in locomotor ataxia) and of the *skin* (such as acute decubitus and "glossy skin") are apparently connected in some way with those nerves and centers which are concerned with sensation. The ganglia on the posterior roots of the spinal nerves are frequently found diseased in cases of zona.

Lesions of the peripheral nerves will occasion nutritive disturbances like those produced by spinal lesions.

Irritative lesions of nerves cause much more severe trophic disturbances than *section* of a nerve would cause.

Disturbances of the Electrical Reactions of Nerves and Muscles.

The normal reactions are—

1. With weak currents = $KaSz - KaO - AnS - AnO$.
2. With moderate currents = $KaSZ - KaO - AnSz - AnOz$.
3. With very strong currents = $KaSTe - KaOz - AnSZ - AnOZ$.

(Ka = Cathode; An = Anode; S = Closing; O = Opening; z = slight contraction; Z = strong contraction; Te = Tetanic contraction.)

The changes due to disease may be (1) quantitative (increase or diminution), and (2) qualitative.

The changes which occur in the electrical reactions of nerves and muscles which have been separated from their trophic centers and are undergoing degeneration, constitute the "*Reaction of Degeneration*," and are as follows:

1. If a *faradaic* or *galvanic* current is passed through a *nerve* which is undergoing degeneration in consequence of either separation from its trophic center or inflammation of the nerve itself, there will be either no response at all or a very feeble one.

2. If a *faradaic* current be passed through a *muscle* which is undergoing degeneration there will be no response, or a feeble one, but—

3. If a *galvanic* current be passed through such a *muscle* the response will be *increased*, actually or relatively, and will show the following changes in character—

- (1) the muscular contractions will be slow, wormlike and persistent, and

- (2) the contractions at the anode will be as marked as those at the kathode, the formula being—

$KaSZ - KaO - AnSZ - AnOZ$.

Diagnostic Value of the Reaction of Degeneration.—This reaction always shows that the lesion is in the large multipolar cells of the anterior cornua or in the peripheral nerves.

Time of Appearance of the Reaction of Degeneration.—The reaction of degeneration appears in three or four days in cases of *irritative* lesions of nerves or trophic centers and in about a week or ten days in cases of *section* of a nerve.

Duration of the Reaction of Degeneration and the Ultimate Result.—The reaction of degeneration usually lasts eight or ten weeks and may terminate either in recovery of normal reaction or in complete loss of all response.

SIMPLE AND MULTIPLE NEURITIS.

Definition and Frequency.—A comparatively rare disease, characterized anatomically by more or less inflammation and degeneration of the nerves and muscles and clinically by pain and paresis in the region of distribution of the affected nerves and by diminution or loss of reflexes, trophic disturbances and the reaction of degeneration.

Causes.—1. *Sex and age.* Women who have passed middle life are most liable to have multiple neuritis

2. *Injuries* of a nerve may cause a simple neuritis.

3. *Exposure to cold*, as in facial paralysis and sciatica.

4. *Certain poisons*, such as *alcohol* (which stands preëminent as a cause of multiple neuritis), lead, arsenic and mercury. *Gout* and *rheumatism* probably act by the retention of morbid matters in the blood in these diseases.

5. *Certain infectious diseases*, such as kakke (or beri-beri), diphtheria, anæsthetic leprosy and typhoid fever.

Morbid Anatomy.—1. *Extent.* One or many nerves may be involved. In the simple form, due to injury, a single nerve is usually affected.

In multiple neuritis many nerves are implicated, especially the musculo-spinal and the anterior tibial. The corresponding nerves on the two sides are usually affected.

2. *Changes in the nerves.* There may be simply inflammation with serous exudate in the nerve sheath, or the interstitial connective tissue and even the nerve fibres may be involved. In old, standing cases the connective tissue is increased and the nerve fibres undergo Wallerian degeneration.

3. The *muscles* also undergo degenerative changes, the striations disappearing and the fibres becoming small and granular.

4. Sometimes the *skin* undergoes an atrophic change and becomes smooth and shining ("glossy skin").

Symptoms.—1. *Sensory.* Pain and tenderness along the nerve and at its distribution during the early stages of the disease from the increased blood supply to the nerves; various forms of *paresis* occur also, such as numbness, tingling, &c. At a later stage *anæsthesia* occurs from destructive changes in the nerve fibre.

2. *Motor.* In the early stages motion is little affected, except in severe cases, but when the disease is interstitial and the nerve fibres degenerate there is *paræsis* or, possibly, complete *paralysis*.

Ataxia is sometimes a marked symptom.

3. *Reflexes.* In the very early stages the reflexes are increased from the increased blood supply, but later they are impaired in all cases, from loss of conducting power in the degenerated nerves and consequent interruption of the reflex circuit.

4. *Trophic*. Atrophy and degeneration of muscles, herpes, œdema and glossy skin are of common occurrence.

5. *Electrical reactions*. The reaction of degeneration or the *partial* reaction of degeneration is present in nearly all cases.

In the primary forms of multiple neuritis, due to alcohol or to infectious diseases, the

6. *Onset* is sudden, as a rule, and

7. The *temperature* is elevated— 102° to 104° .

Diagnosis.—1. From *anterior poliomyelitis* by the absence of sensory disturbances in the latter.

2. From *loco-motor ataxia* by the absence of eye symptoms, the rapid onset and the presence of the reaction of degeneration in multiple neuritis.

Course and Prognosis.—The *course* is usually slow, but in some cases of multiple neuritis there may be a rapidly fatal course; the *prognosis*, however, is usually favorable.

Treatment.—1. To *relieve pain* by rest, phenacetine, the salicyl compounds and morphia. No form of electricity is advisable in acute cases. *Hot* applications may cause blisters or even sloughing.

2. In the *late* stages to promote absorption and stimulate nutrition by iodide of potassium, arsenic, cod liver oil, strychnia, galvanism (and possibly faradism) and massage.

FACIAL PARALYSIS OF PERIPHERAL ORIGIN.

Definition and Frequency.—A common form of paralysis involving the muscles of one side of the face and due to disease or injury of the facial nerve on the distal side of its nucleus.

Causes.—1. *Cold* and *exposure to draughts*, which probably cause neuritis.

2. *Caries of petrous portion* of the temporal bone, the inflammation in this case extending to the nerve and causing neuritis.

3. *Pressure* on the nerve by tumors at the base of the skull or by pieces of bone in fracture of the base of the skull or, rarely, by tumors of the parotid gland.

4. Injuries which directly involve the facial nerve.

Symptoms.—The *symptoms* vary according to the degree of degeneration of the nerve.

1. The *onset* may be gradual, but the paralysis often occurs in a single night.

2. *Sensory* disturbances are entirely absent.

3. *Motor*. There is a paralysis of the orbicularis of the eye and all the muscles on one side of the face; the eye on that side remains open, the mouth is drawn to the opposite side and the normal furrows in the cheek are lost on the paralyzed side. *Mastication* is difficult, because the buccinator is paralyzed, and the food accumulates in the cheeks.

4. *Reflexes and Electrical Reactions*. The reflexes are impaired or lost and the reaction of degeneration is present in all but very mild cases.

Diagnosis.—The *diagnosis* is very easy. The muscles which are paralyzed show that the facial nerve is involved and the reaction of degeneration shows that the disease is either peripheral or in the facial nucleus, but if the *facial nucleus* is involved (as in labio-glosso-laryngeal paralysis) there is more marked atrophy and the paralysis is not limited to the facial nerve.

Course, Duration and Prognosis.—The course of facial paralysis is usually slow; rarely recovery occurs in two or three weeks, but usually it lasts several months and occasionally it is permanent. There is no danger to life. If the electrical reactions remain normal it shows that only the nerve sheath is involved and recovery will probably occur at a comparatively early period.

Treatment.—1. To *remove the cause*, such as syphilis, inflammation of the ear, &c.

2. After the *acute stage* has passed, to stimulate the nerves and muscles by electricity (especially *galvanism*) and massage. *Strychnia* is of service in old standing cases.

All Peripheral Paralyses are similar with respect to their Causes, Morbid Anatomy, Course and Prognosis and Treatment.

The Symptoms and Diagnosis are easily understood from a consideration of the distribution and function of the different nerves.

NEURALGIA.

Definition and Frequency.—A very frequent affection, characterized by violent pain, paroxysmal in character, usually sudden in

occurrence and not due to any morbid anatomical change in the nerves or other tissues.

Causes.—A. *Predisposing*.—1. *Age*. The disease is rare in early childhood, and is most common in young adults.

2. *Sex*. As a rule, women are rather more liable to most forms of neuralgia than men are; sciatic neuralgia, however, is more common in men.

3. *A neuropathic tendency*.

4. *Impaired nutrition*, especially anæmia.

B. *Exciting*. 1. *Cold* is a very common cause, especially of facial neuralgia.

2. *Mechanical irritation*, as by a decayed tooth, the pressure of a tumor or of hardened feces on the nerves in the pelvis, &c.

3. *Malaria*, which is also a predisposing cause by interfering with nutrition, often seems to be a direct exciting cause, the paroxysms of pain occurring at definite periods.

4. *Certain morbid matters* in the blood, such as lead, zinc, uræmic solids, sugar, &c.

5. Irritations *reflected* from more or less distant parts, as in the case of sciatica, caused by stricture of the urethra, &c.

Morbid Anatomy.—In neuralgia proper there is no morbid anatomical change, but many cases of so-called neuralgia are really neuritis (q. v.).

Symptoms.—1. *Sensory*. Pain is the essential symptom of neuralgia; it is usually limited to the area of distribution of a certain nerve or nerves, is sudden in occurrence (usually), is paroxysmal in character, and is apt to disappear from one place and appear in another. After it has persisted some time tender points appear in the course of the nerve.

2. *Motor and reflex*. There is no actual disturbance of motor power, but movement may cause pain and the reflexes are increased because of the increased irritability of the sensory centers and nerves.

3. *Vaso-motor*. Dilatation of the blood vessels in the painful region occurs, which is probably to be explained by Heidenhain's law that "irritation of a sensory nerve causes dilatation of the vessels in the area to which it is distributed and contraction of the vessels elsewhere."

4. *Secretory*. There is usually increased secretion if the neuralgia involves a mucous membrane or gland, due, probably, to the increased blood supply.

5. *Trophic*. Various disturbances of nutrition have been described in connection with neuralgia, especially when the eye is involved, but a neuritis is probably present in all cases when trophic lesions occur.

6. *Psychical.* Insanity is an occasional complication or sequel of that form of facial neuralgia known as "convulsive tic."

Diagnosis.—The *diagnosis* of neuralgia is based on—

1. Its sudden occurrence.
2. Its paroxysmal character.
3. Its tendency to jump from place to place.
4. The absence of trophic lesions and of the reaction of degeneration.

Prognosis.—The *prognosis* as to life is always favorable. Its *duration* and *recurrence* depend on—

1. *Age*—the prognosis being more favorable in early life.
2. The *cause*. Recovery being prompt and recurrence unlikely if the cause can be removed.
3. The *form* of the disease. Some cases of facial neuralgia in elderly persons being extremely rebellious.

Treatment.—1. To *remove the cause*, such as anæmia, gout, rheumatism, diabetes, malaria, stricture of the urethra, &c.

2. To *relieve pain* by phenacetine, acetanilide, opiates, belladonna, croton-chloral, quinine, gelsemium, aconite, arsenic, electricity, chloroform liniment, chloral-camphor, blisters, &c.

3. To *improve the general health* by iron, arsenic, cod liver oil, strychnine, massage, and nutritious food.

4. *Surgical measures*—

(1) *Nerve stretching* has been tried in sciatica, facial neuralgia and inter-costal neuralgia with variable success; it is only advisable when medical treatment has failed, and if it gives relief, it is usually temporary.

(2) *Nerve section* has been used, especially in facial neuralgia; temporary relief nearly always follows, but it is rarely permanent.

TRIFACIAL NEURALGIA.

(Trigeminal neuralgia, facial neuralgia, tic douloureux.)

Definition.—Neuralgia, involving one or more branches of the fifth pair of cranial nerves; it is the most common form or seat of neuralgia.

Causes.—1. Those of neuralgia in general, with the addition of—

2. Certain *affections of the ear*—otitis media.
3. Certain *affections of the eyes*, especially errors of refraction.

Symptoms.—*Sensory.* Pain, usually very severe, which may involve the whole side of the face and part of the head, or may be limited to one or more branches of the nerves—supra-orbital, infra-orbital and inferior maxillary.

Tenderness also occurs at the points where these branches become superficial.

2. *Motor and reflex.* Reflex spasm of the facial muscles sometimes occurs from the excessive irritability of the sensory centres and nerves.

3. The *secretory* and *vaso-motor* and *trophic* have already been mentioned in connection with neuralgia in general.

Diagnosis.—The *diagnosis* from disease of the antrum and from abscess at the root of a tooth is made by the absence of great tenderness and the intermittent character of the pain.

Course, Duration and Prognosis.—The course and duration vary. As a rule, the attacks are of short duration in young persons, but in elderly persons in feeble health *tic douloureux* is often exceedingly intractable.

Treatment.—The treatment is that of neuralgia in general, but gelsemium is especially useful in facial neuralgia.

SCIATICA.

Definition.—An affection characterized by pain in the region of distribution of the sciatic nerve, and in some cases by extreme tenderness of the nerve itself. In most cases the affection is a neuritis and not a simple neuralgia.

Causes.—1. *Age and sex.* The disease is most common in young adult or middle life.

2. *Pressure* on the nerve by uncomfortable seats, or by tumors, &c.

3. *Stricture of the urethra*—causing a *reflex* sciatica.

4. *Diabetes*, gout and rheumatism are also causes.

Symptoms.—1. *Sensory.* Pain in the region supplied by the sciatic nerve and tenderness in the course of the nerve itself, the tenderness when present being due to neuritis. Paræsthesia in some form is common.

2. *Motor.* In many cases (where there is neuritis) there is more or less paresis of the muscles of the leg.

3. In severe cases where the affection is *neuritic* in character the *reflexes* are impaired, *trophic* disturbances such as œdema and even muscular atrophy occur, and the *reaction of degeneration* is present.

Diagnosis.—True neuralgia is distinguished from *neuritis*, affecting the sciatic nerve by the more paroxysmal character of the pain, the absence of marked tenderness, and of all evidences of degenerative change in cases of simple neuralgia.

Prognosis and Duration.—The *prognosis* is favorable, but the disease often lasts for months, and relapses are frequent.

Treatment.—The *treatment* is that of neuralgia in general. If the case be one of neuritis great care should be exercised in the use of hot applications for fear of causing severe blisters or even sloughing from defective trophic influence. (Gowers.)

Salol and phenacetine give great relief in many cases.

INTER-COSTAL NEURALGIA.

Definition and Frequency.—A very frequent affection characterized by neuralgic pain in the inter-costal nerves.

Causes.—This form of neuralgia is most common in young women who are *anæmic*, and especially if *uterine disease* be present.

Symptoms.—1. *Sensory.* Pain in the region of distribution of one or more inter-costal nerves is the essential symptom. There are tender points near the vertebral column, in the axillary line and in front where branches of the nerves become superficial.

2. *Motor, reflex, trophic* and *electrical* disturbances are only present in cases of neuritis.

Diagnosis.—1. From *neuralgia* it is distinguished by the tender points and more paroxysmal character of the pain in inter-costal neuralgia.

2. From *pleurisy* by the absence of pleuritic friction sounds and of fever.

Prognosis.—The *prognosis* is uniformly favorable.

Treatment.—The *treatment* is that of neuralgia in general.

DISEASES OF THE SPINAL CORD.

ACUTE SPINAL MENINGITIS.

Definition.—An acute inflammation of the membranes surrounding the spinal cord, the pia mater being chiefly involved

Causes.—1. *Age and sex.* The disease occurs most frequently in young men; the reason is not known.

2. *Exposure to intense heat or cold* is a frequent cause.

3. *Injuries*, such as the puncture of a spina bifida or injuries to the spine.

4. *Certain acute infectious diseases*, such as typhoid fever, pneumonia, &c.

5. *Extension* from neighboring parts, as in vertebral caries.

6. *Tuberculosis*, and possibly *syphilis*; tuberculosis is a frequent cause, but the cerebral meninges are usually involved at the same time.

Morbid Anatomy.—1. *Seat and extent.* The pia mater is chiefly affected, and over a considerable extent, because the loose tissue offers no obstacle to the spread of the inflammation.

2. *Condition of the vessels.* The blood vessels are engorged with blood.

3. *Changes in the membranes.* The membranes are reddened, cloudy from albuminoid degeneration of their lining cells, and thickened from the exudate.

4. *Nature of the exudate.* The exudate is serous, sero-purulent or almost purely purulent, but usually contains flakes of fibrin.

5. *Changes in the cord and nerve roots.* The cord in severe cases is more or less inflamed and infiltrated with serum and white blood cells; the nerve roots are also inflamed; the inflammation may be confined to the sheath, or the nerve fibres themselves may be involved.

Symptoms.—The *symptoms* in the early stages are those of *irritation*; in the later stages, those of *depression*.

1. *Sensory.* Hyperæsthesia of the skin and pain on movement are present in the early stages, and are due to the hyperexcitability of the inflamed nerves and nerve centres; later there may be anæsthesia from pressure of the exudate or degeneration of the nerve fibres.

2. *Motor and reflexes.* *Spasms* in the muscles supplied by the nerves coming from the inflamed area is observed, and the *reflexes* are *increased* in the *early stages* from the increased irritability of the nerves and nerve centres. *Later* there may be more or less paresis and weakening of reflexes from pressure on the cord and nerve roots by the exudate, or from degeneration of the nerve fibres.



If the *upper part* of the cord (cervical) is involved, spasm of respiratory muscles occurs, causing *dyspnea*.

3. The *temperature* is elevated— 102° to 104° .

Diagnosis.—1. From myelitis by the absence of spasms and the more marked paralysis of motion and sensation in myelitis.

2. From *tetanus* by the absence of “lock jaw” and the greater elevation of temperature in spinal meningitis.

3. From *rheumatism* by the absence of fever, as a rule, in muscular rheumatism, and the absence also of hyperæsthesia and distinct spasm.

Prognosis.—The *prognosis* is usually unfavorable; it is less grave in *epidemic* cerebro-spinal meningitis than in the form now under consideration.

Complications and Sequelæ.—*Myelitis* is a frequent complication in severe cases, and in case of recovery, which is rare, sclerosis of the cord may occur as a sequel.

Treatment.—1. To *relieve pain* by phenacetine, opiates, bromide, chloral, &c.

2. To *relieve congestion* by cupping, ice bags, warm baths, purgatives, &c.

Chronic Spinal Meningitis is a rare affection; it may follow the acute or may be chronic from the beginning.

The exudate in such cases is fibrinous and productive, and extensive adhesions and thickenings occur.

The treatment consists in the administration of iodide of potassium, mercury and tonics, and the use of electricity, massage and counter-irritation.

CLASSIFICATION OF DISEASES OF THE CORD.

The following classification of diseases of the spinal marrow is commonly employed:

1. *Focal lesions*, in which the disease is confined *primarily* to a *limited part of the long axis* of the cord; such lesions usually involve the whole thickness of the cord, and are consequently often called *transverse* lesions.

Such lesions, however, usually lead *secondarily* to degenerative changes in the *efferent* or motor tracts (crossed pyramidal tracts especially) *below* the seat of lesion and to similar changes in the afferent or sensory tracts (posterior median column, cerebellar tract and antero-lateral ascending tract) *above* the seat of lesion.

2. *Systemic lesions* in which certain definite and distinct *systems* of fibres or cells are involved throughout a great part or the whole

length of the cord, the other portions of the cord being healthy. For example, in spastic spinal paralysis the crossed pyramidal tracts are alone involved, and in infantile paralysis the large multipolar cells in the anterior cornua are exclusively affected.

GENERAL SYMPTOMATOLOGY OF TRANSVERSE LESIONS OF THE SPINAL CORD.

1. *Paralysis of all the muscles below* the seat of lesion, because the cords conveying motor impulses from the brain are divided.

2. *Loss of sensation below* the seat of lesion, because the sensory tracts are divided.

3. *Increased reflexes and muscular rigidity below* the seat of lesion because the inhibitory tracts are divided.

Loss of reflexes opposite the seat of lesion, because the reflex circuit is broken.

4. *Trophic relations and electric reactions normal below* the seat of lesion in muscles, because the trophic centres in the cord are not injured below the lesion.

Degenerative changes and reaction of degeneration opposite the seat of lesion, because the trophic centres there are destroyed.

5. *Functions of the bladder and rectum impaired*, because the sensory tracts are divided and the patient is no longer conscious of the necessity for emptying these organs.

ACUTE AND CHRONIC MYELITIS.

Definition.—An acute or chronic inflammation of the spinal marrow nearly always transverse and limited in extent.

Causes.—Often unknown.

1. Exposure to cold.
2. Exhaustion.
3. Sexual excesses.
4. Syphilis.
5. Acute infectious diseases.
6. Extension from the meningis.

The way in which these causes act is not known except with respect to the last, which is self-evident. The influence of sexual excesses is very doubtful.

Morbid Anatomy.—1. *Extent.* As a rule, the whole of the cord transversely is involved, but the disease extends only a short distance upwards and downwards except along the sensory and motor tracts respectively.

2. *Consistence.* In the *early* stages the cord is softened at the inflamed spot from the serous exudate. In the *late* stages it is hardened because the white cells have become converted into connective tissue.

3. *Color.* The color is redder than natural, and the outline between the white and gray matter is lost.

4. *Structural changes.* The nerve fibres undergo the changes studied under Neuritis, q. v., and there is an infiltration with serum and white blood corpuscles in the *early* stages; later on, if the patient lives, there is a great increase in connective tissue at the inflamed spot, and a corresponding atrophy of nerve fibres.

Symptoms.—The *symptoms* depend on the *seat* of the lesion.

1. *Motor.* There is paraplegia *below* the seat of lesion, which is preceded for a short time by cramps in some cases, from the irritability of the nerves and nerve centres in the cord during the first stage. Fibrillar twitchings occur, which cannot be explained satisfactorily.

2. *Sensory.* Slight and transient hyperæsthesia at first, from the congestion of the nerves and centres in the cord; later *anæsthesia* below the seat of lesion; *opposite* the seat of lesion a "girdle pain," probably due to the congestion at the upper limit of the inflamed spot.

3. *Reflexes and Trophic* (see General Symptomatology).

4. *Urinary and rectal.* These symptoms depend on the seat of lesion; if it is above the lumbar enlargement there is retention of urine and feces (see Symptomatology) but if the lumbar cord and the vesical and rectal centres are involved there is paralysis of the sphincter with incontinence. (Incontinence of urine may occur from overfilling of the bladder also.)

5. *Temperature.* If the cervical cord is affected primarily or by extension there is a marked elevation of temperature, often to 109° or 110° .

6. *Respiratory.* If the cervical cord be involved there is *dyspnoea* from paralysis of the muscles of respiration.

Diagnosis.—1. From *spinal meningitis*. (See Meningitis).

2. From *hysterical paraplegia* by the absence of trophic lesions or reaction of degeneration at any part in hysterical paraplegia and by the history of previous hysterical attacks.

Course and Prognosis.—Myelitis usually runs an acute and fatal course, but more or less chronic cases sometimes occur. Occasionally, especially after injuries to the cord (from fracture of the spine, &c.) an ascending myelitis results.

Complications.—1. *Bed sores* are of common occurrence, from pressure and defective cleanliness.

2. *Cystitis* and *pyelo-nephritis*, from retention of urine or the use of a septic catheter.

Causes of Death.—1. *Asphyxia*, from paralysis of the respiratory muscles.

2. *Prostration* from bed sores, &c.

3. *Pyelo-nephritis*, leading to urinæmic poisoning.

Treatment.—1. To *remove the cause*, such as syphilis, by mercury and iodide of potassium.

2. To *lessen congestion* by warm baths, ergot (?), position, &c.

3. To *prevent cystitis* by using a clean catheter.

4. To *prevent bed sores* by cleanliness and the use of a water-bed and to treat them when formed by iodoform and Peruvian balsam, &c.

5. To *relieve other complications*, such as cystitis, constipation, &c.

6. To *stimulate nutrition of the cord*, after the acute stage, by galvanism, strychnine, arsenic and other tonics.

DISSEMINATED SCLEROSIS.

(Insular sclerosis. Sclerosis in patches.)

Definition and Frequency.—A rather rare disease, characterized *anatomically* by the formation of patches of connective tissue in different parts of the brain or cord, and clinically by various nervous symptoms, of which intention tremor, scanning speech and nystagmus are the most prominent.

Causes.—Very little is understood as to the causes. Men between twenty and forty are usually affected. *Heredity, exposure, overwork* and *syphilis* are possible causes.

Morbid Anatomy.—1. The *extent* is variable; the *size* of the patches varies from that of a pea to that of a small hickory nut; the most common *seat* is in the gray matter of the cord or the white matter of the cerebrum; the lateral ventricles and pons are frequently affected.

2. The *membranes* over the spots are thickened.

3. *Structural changes.* The spots consist of connective tissue, cells and degenerated nerve fibres.

Symptoms.—The symptoms depend on the seat.

1. The *onset* is usually gradual.

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2. *Motor*. Tremor at every attempt to move is present in nearly all cases; if the motor tracts are involved paralysis may occur. Epileptiform attacks are common.

3. *Sensory* symptoms are absent as a rule.

4. *Speech*. The speech is slow, deliberate and "scanning" in character.

5. *Ocular*. Nystagmus is present in nearly all cases.

6. *Mental*. Patients with this disease are very emotional. Apoplectic attacks, with loss of consciousness, occur in the later stages.

No explanation of the tremor, character of the speech and nystagmus has been found.

Diagnosis.—1. From *paralysis agitans*, by the *constant* tremor in the latter.

2. From *locomotor ataxia* by the greater excursion of the movements in the latter disease and by the condition of the reflexes.

Prognosis and Duration.—The *prognosis* is bad and the duration usually many years.

Treatment has been useless thus far—nerve tonics, galvanism nitrate of silver, chloride of sodium and gold, and other agents, have been tried without success.

LOCOMOTOR ATAXIA.

Definition, Synonyms and Frequency.—A disease of the spinal marrow, characterized anatomically by sclerosis of the posterior columns of the cord and clinically by lightning pains, loss of certain reflexes and loss of coördinating power of the muscles. It is one of the most frequent of the spinal affections.

Causes.—1. *Age and sex*. Men between twenty and fifty are most liable to the disease; it is rare in childhood and rarely begins after fifty.

2. *Depressing conditions*, such as overwork, sexual excesses, &c., are said to be causes.

3. *Heredity* (or the hereditary neuropathic tendency) is said to be a cause.

4. *Syphilis* is a cause in a large percentage of cases.

Morbid Anatomy.—1. The *spinal meninges* over the posterior columns are thickened and opaque.

2. The *posterior columns* of the cord, especially the columns of

Gall are *sclerosed*: there is a great increase of connective tissue with corresponding destruction of nerve fibres.

3. The *posterior nerve roots* are more or less degenerated.

Symptoms.—1. *Sensory.* *Lightning pains* in the legs usually occur very early, *numbness* (in the feet especially), *delayed sensation*, and attacks of gastric pain ("gastric crises").

Generative, vesical and rectal. Loss of sexual power, difficulty in emptying the bladder and constipation result from interference with the reflexes.

3. *Ocular.* Argyll-Robertson's pupil (see Reflexes) atrophy of the optic nerve and sometimes paralysis of certain muscles of the eye occur early.

4. *Reflexes.* *Absence of patella tendon reflex* and failure of the pupil to respond to light, though it changes for near and far vision, occur early and are of diagnostic import.

5. *Motor.* No loss of motor power, but *loss of coördinating power*, especially in the lower limbs.

6. *Trophic.* Inflammatory affections of the *joints* occur in a considerable proportion of the cases at a later stage, and in a few cases multiple fractures of bones have been observed (Charcot.)

Diagnosis.—1. From *cerebellar disease* by the absence of sensory symptoms and the giddiness in the latter affection.

2. From *chorea* by the occurrence of bizarre movements in chorea when no attempt at voluntary movement is made.

3. From *general paresis* by the absence of cerebral symptoms.

4. From *multiple neuritis* by the absence of the reaction of degeneration.

Course and Prognosis.—The *course* of locomotor ataxia is very slow, the disease often lasting for years, but recovery is practically unknown.

Treatment.—A. *Hygienic.* The avoidance of fatigue and of cold is important.

B. *Remedial.* No principles of treatment can be laid down in this disease. Mercury and the iodides have not given the results which were expected from them; nor have nitrate of silver, zinc and chloride of sodium and gold, phosphorus, cod-liver oil and galvanism.

Nerve stretching (of the sciatic) has seemed to ameliorate the symptoms.

Suspension relieves the lightning pains in many cases, but does not exert a curative influence.

In all stages of the disease phenacetine, acetanilide or antipyrine are useful to allay the lightning pains or gastric crisis.

SPASTIC SPINAL PARALYSIS. (Spasmodic Tabes Dorsalis).

Definition and Frequency.—A chronic disease of rare occurrence, characterized anatomically by *sclerosis* of the *lateral columns* of the spinal cord and clinically by gradually progressive *paresis* and great *increase of reflexes*.

Causes.—The causes are unknown. The disease is most common in *young men* and has been attributed to overwork and exposure to cold. Syphilis is a probable cause in a few cases.

Morbid Anatomy.—The *spinal cord* only is affected; there is a *sclerosis of the crossed pyramidal tracts* in the lateral columns of the cord on both sides. The nerve fibres atrophy and disappear and there is a great increase of connective tissue.

Symptoms.—1. The *onset* is gradual and the *course* progressive.

2. *Motor.* There is a gradually increasing *paresis* on both sides—the legs being primarily and often exclusively involved; the paresis is due to a gradual destruction of the motor-conducting cords in the pyramidal tracts.

3. *Reflexes.* The reflexes are greatly increased, so that any attempt to move brings on a spasm of the muscles of the leg; the increase of the reflexes is due to the destruction of the inhibitory-conducting fibres in the lateral columns.

4. *Sensory, trophic* and *electrical* disturbances are never present in uncomplicated cases.

Diagnosis.—1. From *loco-motor ataxia* by the great increase of reflexes and the absence of sensory disturbances.

2. From *anterior poliomyelitis* by the absence of muscular atrophy and the increase of the reflexes.

3. From *amyotrophic lateral sclerosis* by the occurrence of muscular atrophy in the latter disease.

4. From *multiple sclerosis* by the absence of intention tremor. If patches of sclerosis involve both lateral columns, however, the symptoms of spastic spinal paralysis will be present (Charcot).

Course, Duration and Prognosis.—The *course* of the disease is usually progressive, the *duration*, months or years, and the danger to life very slight.

Treatment.—Galvanism, nerve tonics and nerve stretching have been tried without benefit. Massage and prolonged warm baths have seemed to lessen the spasm. Strychnine is always injurious,

AMYOTROPHIC LATERAL SCLEROSIS.

Definition and Frequency.—A rare disease of the spinal cord, chronic in course and characterized anatomically by sclerosis of the crossed pyramidal tracts and atrophy of the large multipolar cells in the anterior cornua of gray matter.

Causes.—The causes are unknown; the disease is most common in *young men*.

Morbid Anatomy.—1. *Cord.* The *pyramidal tracts are sclerosed*—the nerve fibres being degenerated and atrophied and the connective tissue greatly increased and the large *multipolar cells in the anterior cornua are atrophied*. The upper part of the cord is affected first. At a late stage the nuclei of the hypoglossus and vagus accessory become involved.

2. *Muscles.* The muscles corresponding with the diseased part of the cord are degenerated and atrophied.

Symptoms.—1. *Motor.* There is a gradual loss of motor power from two causes—first, because the conducting cords in the pyramidal tracts are gradually destroyed, and secondly, because of the degeneration of the muscles themselves.

2. *Trophic.* The motor *nerve fibres* connected with the atrophied cells in the anterior cornua and the muscles which they supply are degenerated because their trophic cells are destroyed. The paresis and atrophy begin in the muscles of the hand and arm, the legs are subsequently affected and still later the muscles of speech and deglutition, because the nuclei in the medulla undergo degeneration.

3. *Reflexes.* The *reflexes are increased* in spite of the degeneration of the multipolar cells, because some of the reflex circuits remain and inhibitory impulses from Setschenow's center are cut off by the sclerosis of the pyramidal tracts.

4. *Electrical.* In the degenerated muscles there is complete or partial reaction of degeneration.

5. *Sensory* disturbances are absent.

Diagnosis.—1. From *spastic spinal paralysis* by the absence of any muscular degeneration in the latter.

2. From *progressive muscular atrophy* by the absence of spastic symptoms in this disease.

Course, Duration and Prognosis.—The *course* is progressive, the *duration* long, the *prognosis*, as to recovery, hopeless.

Treatment.—The *treatment* is purely symptomatic and is utterly ineffectual.

CHRONIC BULBAR PARALYSIS.

(Glosso-labio-laryngeal paralysis.)

Definition.—A chronic disease of comparative rarity, characterized anatomically by atrophy of certain nuclei in the medulla oblongata, and clinically by gradually progressing paresis and atrophy of the muscles of the tongue, lips, pharynx and larynx.

Causes.—The causes are unknown; the disease is more common in men after 35 years of age.

Morbid Anatomy.—1. *Medulla oblongata.* There is degeneration and atrophy of the nuclei of the *hypoglossus*, *vagus*, *accessory*, *facial* and *glosso-pharyngeal* nerves.

2. The *nerves* leading from them are degenerated and also the *muscles* which they supply, namely—the tongue, the muscles of the lips, pharynx and larynx.

Symptoms.—1. The *onset* is very gradual; (rarely there is an acute form of bulbar paralysis.)

2. *Motor and trophic.* There is gradual *loss of motor power and simultaneous degeneration* and atrophy of the *tongue and lips* leading to difficulty in *speech*, of the *pharynx*, leading to difficulty in *swallowing* and consequent emaciation, and of the *larynx*, leading to *respiratory disturbances* and to secondary bronchitis and pneumonia from the entrance of foreign bodies into the air passages.

3. The *reflexes* (throat reflex especially) are lost or greatly diminished from the break in the circuit (atrophy of cells, nerves and muscles). Occasionally reflexes are increased from simultaneous atrophy of the inhibitory fibres above the nuclei in the medulla.

4. The *pulse* is sometimes very rapid from involvement of the vagus center.

5. *Sensory* symptoms are absent.

Diagnosis.—The *diagnosis* is based on the paresis or paralysis and muscular atrophy in the region supplied by the bulbar nerves.

Course, Duration and Prognosis.—The *course* is progressive, the *duration* from two to five years, the *prognosis* hopeless.

Causes of Death.—The causes of death are—

1. *Exhaustion* from inability to swallow.
2. *Asphyxia* from laryngeal paralysis.
3. *Cardiac failure* from involvement of the vagus.
4. *Complications*, such as in inhalation pneumonia.

Treatment is without avail. Galvanism, nitrate of silver, ergot and many other remedies have been tried; none have been found of much, if any, service.

ACUTE ANTERIOR POLIOMYELITIS. (Infantile Paralysis.)

Definition and Frequency.—A disease of comparatively common occurrence in children, rare in adults, acute in course, characterized anatomically by degeneration of the large multipolar cells in the anterior cornua of gray matter of the cord, of the nerves passing out from these cells and the muscles which these nerves supply, and characterized clinically by muscular atrophy and paralysis of certain muscles or groups of muscles.

Causes.—1. *Age*. The disease is far more common in children between the ages of one and four years than at any other age.

2. *Season*. It occurs chiefly in hot weather, and is probably due to chilling when overheated in some cases (Gowers).

3. There are many symptoms which suggest that the disease is *infectious* in character.

Morbid Anatomy.—1. *Cord*. There is degeneration and atrophy of the large cells in the anterior cornua and an increase of the connective tissue at the same point.

2. *Nerves and muscles*. The *nerves* passing from these cells to the muscles, and the muscles themselves are degenerated.

3. The *bones* often fail to keep pace in growth with other parts of the body and the *joints* are loose and flabby, probably in great part from the loss of muscular support.

Symptoms.—1. *Onset*. The onset is sudden.

2. *Temperature*. The temperature in the early stage is elevated (103° — 105°) and this may continue several days.

3. *Nervous and muscular*. Convulsions, unconsciousness or delirium are frequent in the early stages. Later the characteristic symptoms appear, which are—

4. *Motor and trophic*. There is paralysis in one or more limbs, with wasting of the muscles from atrophy of the trophic cells in the anterior cornua.

5. *Reflexes*. The reflexes are lost, because the circuit is broken.

6. *Electrical*. There is *reaction of degeneration*, because of degenerative change in the muscles and nerves.

7. *Sensory*. There is *no sensory* disturbance, because the disease is confined to motor and trophic cells and nerves.

Diagnosis.—The disease is often confounded with *Neuritis* (Gowers) from which it may be distinguished by the absence of sensory disturbances in poliomyelitis.

Prognosis.—The *prognosis* as to life is good in the great majority of cases, and in nearly all cases there is great *diminution* also in the *extent of paralysis* in the course of a few weeks, but atrophy of the muscles of one limb is *usually permanent*. Complete recovery may occur, but is rare.

Treatment.—A. In the *early* stage the *bromides* and *phenacetine* or *acetanilide* to reduce fever and nervous irritability.

B. At a *later* stage *galvanism* to stimulate regeneration of the nerves, *strychnine* and possibly *ergot* (?) for the same purpose, and *mechanical appliances* to correct the resulting deformities (talipes, &c.)

PROGRESSIVE MUSCULAR ATROPHY.

Definition and Frequency.—A rather rare disease, very gradual in its onset, chronic in its course and characterized by atrophy of the large cells in the anterior cornua and of certain muscles, with consequent paralysis.

Causes.—The causes are very obscure.

1. *Age and sex.* Young adults are more liable to it than persons at other periods of life and men more than women.
2. *Excessive work of certain muscles* (?)
3. *Heredity and consanguineous marriages* (?)

Morbid Anatomy.—1. *Cord.* The large cells in the anterior cornua are degenerated and the connective tissue around them is increased. There is no other morbid change in the spinal cord.

2. The *anterior* nerve roots and motor *nerves* and the *muscles* are also degenerated.

Symptoms.—1. The *onset* is exceedingly gradual.

2. *Motor and trophic.* Atrophy and paralysis of certain muscles; the thenar and hypo-thenar eminences are nearly always involved first, then the other muscles of the hand, of the arm and of the trunk; the thigh and leg muscles are involved last.

3. The *reflexes* are diminished or lost because of the changes in the motor nerves and nerve cells.

4. The *reaction of degeneration* or the *partial reaction of degeneration* is *present*.

Sensory symptoms are *absent*.

Diagnosis.—1. From *neuritis* by the absence of sensory disturbances.

2. From *amyotrophic lateral sclerosis* by the absence of spasm.

3. From *acute anterior poliomyelitis* by the gradual onset and absence of fever.

Prognosis and Duration.—The disease is chronic in course, of *long duration*, often fifteen or twenty years, but is hopeless as to recovery.

Treatment is of no avail.

PSEUDO-HYPERTROPHIC PARALYSIS.

(Primary Muscular Atrophy.)

Definition and Frequency.—A rare disease, characterized by atrophy and paralysis of some of the muscles without any affection of the cord.

Causes.—1. *Age and sex.* Boys between eight and twelve years old are usually affected.

2. *Heredity.* Several members of the same family may be sufferers with the disease.

3. *Neuropathic tendency.*

Morbid Anatomy.—1. *Muscles.* The muscles of the back and thighs are usually involved. The fibres are small, but *not degenerated* nor granular, nor do they lose their striature. In some cases there is an apparent hypertrophy of the muscles from an interstitial deposit of fat.

2. There is *no change in the nervous system.*

Symptoms.—1. *Motor.* Gradual loss of motor power in the muscles of the back and thighs and later in other muscles.

2. The *reflexes* are diminished because of the loss of muscular tissue (*simple atrophy*).

3. *No reaction of degeneration*, because no degeneration occurs.

4. The *trophic* lesions consist in simple atrophy of muscles and usually an increase in connective tissue and interstitial fat.

Diagnosis.—From *progressive muscular atrophy* by the different muscles involved, the age of the patient and the absence of reaction of degeneration.

Prognosis.—The *prognosis* is unfavorable; the *course* is slow.

Treatment.—*Electricity* and *massage* are said to have caused some temporary improvement, or, at least, to have checked, for a time the progress of the disease.

No other agents have produced any effect.

ACUTE ASCENDING PARALYSIS.

(Landry's Paralysis.)

Definition and Frequency.—A rare disease, characterized by a rapidly developing paralysis, commencing in the lower limbs and extending upwards, with elevation of temperature and splenic enlargement.

Causes and Morbid Anatomy.—The *causes* and *morbid anatomy* are unknown. It is probable that in some cases the disease is a multiple neuritis and in others an acute ascending spinal paralysis. In many respects the affection resembles the *acute infectious* diseases.

Symptoms.—1. *Motor*. Paralysis usually commencing in one lower limb, involving the other in a short time and rapidly extending upwards.

2. *Sensory*. The sensory symptoms are variable, often there are none; sometimes there is anæsthesia.

2. *Reflexes* usually lost.

3. *Electrical* reactions variable.

5. *Fever*—(102° or 103°)—and slight splenic enlargement in all cases.

Diagnosis.—The *diagnosis* is based on the course and character of the symptoms.

Prognosis and Duration.—Duration a few weeks. Many cases end in recovery.

Treatment.—Symptomatic in *early* stages.

Electricity and massage later.

DISEASES OF THE BRAIN.

CEREBRAL HYPERÆMIA.

Definition and Varieties.—An excess of blood in the brain which may be—1. *Active*, when an excess of arterial blood reaches the brain, and 2. *Passive*, when there is some obstacle to the return of venous blood from the brain.

Causes.—A. Of the *active* form—1. *Alcohol*, which causes increased action of the heart and dilatation of the vessels.

2. *Certain drugs*, as amyl nitrite, &c., which cause dilatation of the cerebral vessels.

3. *Over exertion*, which increases the force and frequency of the heart's action.

B. Of the *passive* form—Any obstruction to the venous circulation, such as occurs in the later stages of cardiac disease, and from pressure of tumors in the neck, and, also, any serious interference with respiration, such as occurs in croup.

Morbid Anatomy.—The *morbid anatomy* is not striking—some dilatation of the vessels, with increased redness of the brain and membranes, and in the passive form some serous effusion is all that is found.

Symptoms.—A. In the *active* form *increased mental activity, sleeplessness, hyperæsthesia, &c.*, from increased cerebral activity.

B. In the *passive* form, *depression* of all the cerebral functions, drowsiness, stupor, &c.

Diagnosis.—The diagnosis is difficult; it is based on the character of the symptoms, the causes and the absence of any evidence of organic disease.

Prognosis.—The *prognosis* depends upon the cause; it is usually good in the active form and serious in the passive.

Treatment.—To reduce the amount of blood in the brain—

1. In the *active* form by bromides, cold to the head, intestinal derivation, counter irritants, &c.

2. In the *passive* form by digitalis, ergot, &c.

CEREBRAL ANÆMIA.

Definition and Varieties.—A diminution of the amount of blood in the brain; it may be—1. General, or 2. Local.

The general form only will be described here.

Causes.—1. *General anæmia.*

2. *Certain cardiac diseases*, such as aortic obstruction and failing compensation in any form of heart disease.

3. *Reflex* from disturbances of the stomach or other organs.

4. *Cerebral*, as mental shocks.

Morbid Anatomy.—More or less pallor of the brain and its membranes.

Symptoms.—In *acute* cases (an ordinary *fainting fit*), giddiness, nausea, pallor, loss of consciousness and loss of motor power and sensation; such an attack usually lasts only a few moments.

In *chronic* cases, want of decision of character, irritability of temper, pallor and the other symptoms of the causative condition.

Diagnosis.—The *diagnosis* is based on the symptoms and history of the case and presents no difficulty.

Prognosis.—The *prognosis* of an ordinary fainting fit is nearly always good.

The *prognosis* of the chronic form of cerebral anæmia depends on the cause: in cases of general anæmia it is usually good, in cardiac diseases unfavorable.

Treatment.—1. Of a *fainting fit*—to increase the amount of blood in the brain by the *horizontal position*, *amyl nitrite*, alcoholic stimulants and ammonia.

2. Of the *chronic form*—to remove the cause, if possible, by iron, strychnine and, in most cases, by the use of *digitalis* (in failing compensation).

CEREBRAL MENINGITIS.

Definition and Divisions.—Cerebral meningitis is an inflammation of the membranes surrounding the brain.

1. *Pachymeningitis* is an inflammation of the outer membrane or dura mater. Either the external or internal surface of the dura may be involved, constituting *external* pachymeningitis and *internal* pachymeningitis, respectively.

External pachymeningitis belongs in the domain of surgery.

2. *Simple acute meningitis* or *leptomeningitis* is the form of meningitis which occurs sometimes in connection with injuries, ear diseases, pneumonia, &c.

3. *Tubercular meningitis* or *basilar meningitis* (so called because the base of the brain is chiefly involved) is due to the bacillus tuberculosis.

INTERNAL PACHYMENINGITIS.

(Hæmatoma of the dura mater).

Definition.—A disease characterized by the formation of connective tissue with repeated extravasations of blood on the internal surface of the dura.

Causes.—1. *Age and sex.* The disease is most common in *old men*.

2. *Intemperance.*

3. *Chronic, renal and cardiac disease.*

4. *Syphilis.*

It is not clear how these causes act, but probably in part at least by causing a degenerative change in the walls of the blood vessels.

Morbid Anatomy.—On the internal surface of the dura, usually in the parietal region, there is a new formation of very vascular connective tissue with extravasations of blood of various dates.

Symptoms.—1. The *onset* is usually sudden and *relapses* are frequent.

2. *Mental.* Stupor and coma immediately after the attacks and mental weakening in the intervals, probably due to pressure.

3. *Motor.* Sometimes *convulsive attacks*, from irritation, may occur, but usually there is monoplegia or paresis.

4. *Sensory* disturbances are absent, as a rule.

Diagnosis.—The *diagnosis* is based on the repeated occurrence of the attacks, with mental weakness.

Prognosis.—The *prognosis* is uniformly unfavorable, but life may be prolonged for some time.

Treatment.—The avoidance of stimulants, over exertion and excitement in order to prevent hemorrhage.

The use of iodide of potassium and mercury has been recommended, but has not been followed by any appreciable results.

SIMPLE ACUTE MENINGITIS.

(Leptomeningitis.)

Definition.—An inflammation of the cerebral pia mater.

Causes.—1. *Injuries*, such as fractures of the skull, &c.

2. *Diseases* of the ear, leading to inflammation of the petrous portion of the temporal bone or of the mastoid cells, which, by extension, leads to meningitis.

3. *Certain acute infectious diseases*, especially pneumonia and erysipelas.

Morbid Anatomy.—1. The *pia mater* loses its transparency and becomes cloudy from albuminoid degeneration of its cells and serous infiltration; it is, of course, greatly reddened, and the injected vessels can be seen coursing through the thickened and opaque membrane.

2. The *brain* is injected, and if the exudate is considerable the convolutions are flattened and softened by the serous exudate.

3. The *exudate* is considerable in quantity, is most abundant along the fissures and large vessels, and is sero-purulent in character.

Symptoms.—The symptoms are first, those of *irritation*, and later, those of *depression*.

1. *Sensory*—headache, intolerance of light, hyperæsthesia from the increased irritability of the nerves and nerve centers in the early stages.

2. *Mental*. Delirium in the early stages, followed by stupor and coma from pressure of the exudate.

3. *Motor and reflexes*. *Spasm* of certain muscles, especially those of the eye, in the early stages, with *contraction of the pupil*, from increased reflex irritability, followed by paralysis from pressure on the nerves or nerve centers by the exudate and dilated and immovable pupils, from pressure on the motor oculi nerve and loss of reflex.

4. The *pulse* is at first full and slow from irritation of the brain; later, it becomes rapid and irregular.

5. The *temperature* is elevated— 100° to 102° —as a rule, but it may be higher, especially just before death.

6. *Digestive*. *Vomiting* is common, and is probably due to the irritation of the 4th ventricle. *Constipation* is the rule.

Diagnosis.—1. From *typhoid fever* by the different temperature curve, the absence of eruption and the presence of paralysis of some of the ocular muscles.

2. From *pneumonia* by the absence of the physical signs of the latter disease.

3. From *uræmia* by the absence of albuminuria.

Prognosis and Duration.—The *prognosis* is almost uniformly fatal and the *duration* only a few days.

Treatment.—1. To lessen the amount of blood in the brain by bromides, purgatives and cold to the head.

2. To relieve pain by morphia and the bromides,

TUBERCULAR MENINGITIS.

(Basilar Meningitis ; acute hydrocephalus.)

Definition and Frequency.—An inflammation of the pia mater, especially marked at the base of the brain, and due to the bacillus tuberculosis.

Causes.—1. The *bacillus tuberculosis*.

2. *Secondary* to tubercular disease elsewhere, as in the lungs or glands especially.

3. *Age.* It is far more common in children than in adults.

Morbid Anatomy.—1. *Seat.* The usual seat is at the base of the brain.

There are *tubercles* on the membranes, especially marked along the blood vessels, and also the changes of leptomeningitis (q. v.).

3. The *exudate* is chiefly serous in character, but it contains a number of cells and some fibrinous flakes.

Symptoms.—A. *Prodromic.* Listlessness, headache and slight fever for a week or two, prior to the development of the attack, are common.

B. *Of the developed attack.*

The symptoms are much the same as those already mentioned under simple meningitis, but *spasm* and *subsequent paralysis* of the *ocular muscles* is more marked because the base of the brain is chiefly affected and the nerves are involved where they emerge from the brain.

Convulsions are very prominent in children, probably because the inhibitory centers are not well developed in early life.

The *temperature* is usually higher than in simple meningitis.

Vomiting is a very common symptom, especially in the early stages ; it is projectile in character.

The *abdomen* is "boat shaped," from contraction of the abdominal muscles.

Emaciation is rapid and *prostration* great.

Diagnosis.—The *diagnosis* is based on (1) the *history* of the case ; (2) the *age* of the patient, and (3) the presence of tubercles elsewhere.

Prognosis.—The *prognosis* is unfavorable ; it is doubtful whether recovery ever occurs.

Treatment.—The prophylactic treatment is that of tuberculosis, in general (q. v.).

The *remedial* treatment consists in the administration of the bromides, chloral, &c., to control the convulsions, and in the relief of other symptoms as they arise.

Iodide of potassium is thought by some to be beneficial.

CHRONIC HYDROCEPHALUS.

Definition.—An accumulation of fluid within the cranium, usually in the ventricles of the brain.

Causes.—1. *Age.* The disease is often congenital or occurs in the early months of life.

2. *Family tendency.* Several children in the same family are frequently affected.

Morbid Anatomy.—1. The size of the head is greatly increased.

2. The *skull bones* are very thin and the sutures and fontanelles widely open.

3. The fluid is very thin, contains a little chloride of sodium and is contained chiefly in the lateral ventricles.

Symptoms.—1. *Appearance.* The head is large and round; the eyes turned down so as to show the sclerotic.

2. *Nervous.* The mental powers are weak, and muscular power impaired.

General. The body and limbs are, as a rule, small and shrivelled.

Diagnosis.—The *diagnosis* is very simple.

Prognosis.—The *prognosis* is bad, and hydrocephalic children rarely live to be more than five or six years old.

Treatment.—No *medical* treatment has given any results.

Aspiration of a small quantity of fluid and strapping may be tried.

TOPOGRAPHICAL DIAGNOSIS IN BRAIN DISEASES.

Only a brief outline of cerebral localization will be given here, and only those affections will be considered which most frequently present themselves to the practitioner.

Direct and Indirect Focal Lesions—1. A *direct* focal lesion is one which causes a *destruction* of the nerve cells or fibres, and is more or less permanent.

2. An *indirect* focal lesion is one which causes a *temporary* disturbance of certain nerve centers or conducting cords from *pressure* or temporary interference with the blood supply.

The symptoms produced by a *direct* focal lesion are more or less permanent; those produced by an *indirect* focal lesion are usually temporary.

LOCALIZATION OF BRAIN DISEASES.

SYMPTOMS.

1. Hemiplegia, with facial paralysis and loss of consciousness, but with general improvement, and, possibly recovery.
2. Same symptoms, but with *late rigidity* on paralyzed side.
3. Hemi-anaesthesia, with loss of consciousness.
4. *Motor* aphasia.
5. Word deafness.
6. *Facial* monoplegia or monospasm.
7. *Brachial* monoplegia or monospasm.
8. Monoplegia or monospasm involving the muscles of the *leg*.
9. Crossed *facial paralysis*.
10. Crossed *ocular paralysis*.
11. Inco-ordination, with *increased* tendon reflex and giddiness.
12. Lateral homonymous hemi-anopsia.
13. "Soul blindness."
14. Difficulty of articulation or of swallowing.

SEAT OF LESION.

Direct focal lesion *near* motor tracts, probably in motor ganglia; *indirect* focal lesion of motor tracts—probably internal capsule.

1. Cortical motor centers, or
2. Corona radiata, or
3. Anterior $\frac{2}{3}$ d of internal capsule.

Posterior third of internal capsule.

Posterior part of third left frontal convolution.

Posterior part of first left temporal convolution.

Lower part of ascending frontal and parietal convolutions.

Middle part of ascending frontal and parietal convolutions.

Upper part of ascending frontal and parietal convolutions and para central lobule.

Lower half of pons Varolii.

Cerebral peduncle.

Cerebellum.

Occipital convolutions, (especially the cuneus) on the fibres between them and the optic chiasm.

Angular gyrus on the left side.

Medulla oblongata.

Irritative and Destructive Lesions.—*Irritative* lesions of the cortex cause *spasm*.

Destructive lesions of the cortex cause paralysis.

Epileptiform attacks with a *visual aura* are due to irritative disturbance in the occipital lobes.

Epileptiform attacks with an *aural aura* are due to an irritative disturbance in the first *temporo-sphenoidal* convolution.

It should be remembered, of course, that cerebral spasm and paralysis are on the *opposite side* of the body from the seat of lesion.

In the case of *crossed facial* and *crossed ocular* paralysis, the *face*

or *ocular muscles*, as the case may be, are paralyzed on the *side of lesion*, while the *limbs* are paralyzed on the *opposite side*.

CEREBRAL TUMORS.

Varieties.—1. Glioma.

2. Sarcoma.

3. Gumma.

4. Tubercle.

5. Cancer.

Causes.—1. *Age and Sex.* Brain tumors, with the exception of tubercle, are most common in middle life and in the male sex.

2. No cause can be assigned for any except *syphilis* and *tubercle*.

Symptoms.—A. *General.* 1. *Sensory.* Headache is almost invariably present, and is usually persistent and severe. Hyperæsthesia over a part of the cranium is occasionally present. Headache is due to intra-cranial pressure.

2. *Mental.* Emotional disturbance, hysterical in character, and mental weakness, are common.

3. *Motor symptoms* depend upon the seat.

4. *Circulatory.* The pulse is usually slow, from increased intra-cranial pressure.

5. *Digestive.* *Vomiting* is a common symptom. It is projectile in character and cerebral in origin.

6. *Ocular.* *Choked disk* is present in nearly all cases from increased intra-cranial pressure, and is of diagnostic importance.

7. *General.* Loss of flesh and strength are common, but not invariable symptoms. They are most marked in cases of cancer and tuberculosis.

B. *Focal symptoms* are both direct and indirect, and vary with the seat of the tumor and the amount of pressure it exerts on surrounding parts. (See Topographical Diagnosis in Brain Diseases).

Diagnosis.—1. From *abscess* by the much more gradual onset, the presence of choked disk and the absence of fever, as a rule, in the case of brain tumors.

2. From *apoplexy* and *thrombosis* by the gradual onset.

3. From *hysteria* by the choked disk.

Course, Duration and Prognosis.—The *course* is progressive, the *prognosis* bad, the *duration* from a few months to several years. Recovery is possible if the tumor be syphilitic.

Treatment.—1. To *remove the cause* by antisyphilitic treatment if there is a *suspicion* of syphilis.

2. To *relieve pain and cerebral congestion* by the bromides, phenacetine, &c.; opium is not advisable, because it increases the amount of blood in the brain.

3. *Surgical treatment* is advisable in all cases when the tumor can be definitely located, is probably circumscribed and can be reached without great risk. The removal of tumors, in cases of spasm, due to irritation of the cortex, and also in cases of epilepsy, &c., has often given favorable results.

CEREBRAL HEMORRHAGE.

(Apoplexy.)

Causes.—A. *Predisposing.* 1. *Disease of the walls of the vessels*, such as *miliary aneurisms*, atheroma and syphilis.

2. *Alcoholism, syphilis, gout and Bright's disease*, which cause impairment of the walls of the blood vessels.

3. *Age and sex.* Men in advanced age are more liable to apoplexy than others, because the vascular walls are degenerated in advanced age.

B. *Exciting.* 1. *Violent exertion.*

2. *Mental excitement.*

3. *Cardiac Stimulants.*

4. *Immoderate eating.*

All these causes act by increasing the force of the heart's beat and the pressure in the vessels.

Morbid Anatomy.—1. *Seat.* Any part of the brain may be involved, but the most usual seat of hemorrhage is in the *central ganglia* and *internal capsule*, because the vessels there are subjected to excessive pressure.

2. *Size.* The size varies from that of a small nut to a mass two or three inches in diameter.

3. *Appearance of the clot.* At first, the clot is dark and soft; later, it becomes firmer, and may either be absorbed, leaving a scar, or it may form a sack containing fluid and surrounded by a wall of connective tissue.

4. *Condition of surrounding brain tissue.* At first, the surrounding brain tissue is ragged and infiltrated with serum and some blood corpuscles; later, it returns in part to its normal condition, and a wall of connective tissue is formed around the remains of the clot.

5. *Descending sclerosis.* If the motor centres or motor tracts are destroyed there is a descending degeneration of the pyramidal tracts in the cord.

Symptoms.—The symptoms depend on the seat and size of the clot.

1. The *onset* is nearly always sudden, but severe headache, numbness, &c., may precede the attack.

2. *Mental.* Loss of consciousness occurs in most cases, but not in all; it is due partly to pressure and partly to anæmia of the cortex. *Mental weakness* occasionally occurs at a late stage.

3. *Motor.* Paralysis—nearly always hemiplegia—with involvement of the face is the most striking symptom. *Rigidity of the muscles* of the limbs on the paralyzed side occurs after a time, and is due to the cutting off of inhibitory influences from the brain.

Conjugated deviation of the eyes—the balls being turned upwards and outwards—"looking towards the lesion" is frequent in the early stages. In *right hemiplegia aphasia* usually occurs.

4. *Sensory.* Hemi-anæsthesia occurs occasionally, and is due to the involvement of the posterior third of the internal capsule.

5. *Circulatory.* At first the *pulse* is feeble from shock, later it becomes full and slow, and still later weak and irregular if a fatal termination is approaching.

6. *Temperature.* The temperature *falls at first*, from disturbance of the heat centres; later it rises, probably from inflammatory disturbance around the clot.

7. *Respiratory.* The respirations are full and slow, often irregular, and stertorous in character, from paralysis of the muscles of the soft palate and cheek.

8. *Reflexes.* The *reflexes* are *at first* lessened or abolished, from shock, but *later* the tendon reflexes are *increased in the paralyzed side* because inhibitory influences are cut off.

9. *Trophic.* "Acute decubitus" (bed sores) occurs early on the buttock of the paralyzed side.

Diagnosis.—1. From *uræmia* by the absence of albuminuria.

2. From *thrombosis* and *embolism* with great difficulty by the absence of the causes of thrombosis and embolism and by the age of the patient—apoplexy usually occurring at a later period of life than thrombosis and embolism.

Course and Prognosis.—An attack of apoplexy does not usually terminate fatally at once.

Improvement in motor power usually occurs, and if the lesion of the motor tracts be *indirect* recovery may occur.

Aphasia may be entirely recovered from in the early years of life especially, the right side of the brain taking on the function of articulate speech.

Rigidity of the limbs on the paralyzed side occurs usually if the motor and *inhibitory* tracts are destroyed.

"*Post-hemiplegic chorea*" occurs in some cases.

Mental weakness may result, but is not frequent except in very advanced life.

Recurrence is probable.

Complications.—1. Bed sores.

2. Inhalation pneumonia.

Treatment—1. To *reduce blood pressure* by absolute rest, amyl nitrite, bleeding, &c.

2. To *draw blood from the head* by cold to the head, purgatives, the bromides, &c.

In *late stages*—

3. *Galvanism* (a weak current) to the head—of doubtful value—and faradism and massage to the limbs to prevent contraction of the muscles.

THROMBOSIS AND EMBOLISM OF THE CEREBRAL VESSELS.

Definition and Causes.—(See General Pathology.)

Morbid Anatomy.—1. *Usual seat.* The *left middle cerebral artery* is the usual seat, but other vessels may be plugged.

2. *Changes in the brain substance.* The blood supply to the area of distribution of the plugged vessel is cut off, and *softening* occurs because the vessel is a *terminal* one. The *color* of the softened spot may be either white, red, or yellow. It is *white* in the early stage from coagulation necrosis, or it may be tinged *red* by blood, and later it becomes *yellow* from fatty degeneration. The softened spot contains white blood corpuscles, large corpuscles containing fatty matter, broken up nerve cells and fibres, and granular matter.

Symptoms.—The *symptoms* differ but little from those of cerebral hemorrhage.

The *onset* is often rather more gradual, but this is practically the only difference and not always observable.

Diagnosis.—The *diagnosis* from cerebral hemorrhage is often impossible, but is based on the following points:

1. The presence of *cardiac disease* which would cause embolism.

2. The *age* of the patient. Apoplexy occurs usually in advanced life.

3. The more *gradual onset* (occasionally) of the symptoms of thrombosis.

Prognosis.—The *prognosis* is usually unfavorable, but if the focal lesion does not involve *directly* the motor centres or tracts, recovery may occur.

Aphasia is often more pronounced in embolism and thrombosis than in hemorrhage, but as the disease occurs chiefly in early life recovery from the aphasia is more apt to occur.

Treatment.—The *treatment* does not differ essentially from that of apoplexy, except that bleeding is never advisable in cases of thrombosis and embolism, and sustaining treatment is more often indicated.

Carbonate of ammonia has been advised with a view of dissolving the clot, but its value is exceedingly problematical.

ACUTE CEREBRAL PARALYSIS OF CHILDREN.

(Acute Encephalitis of Children.)

Definition and Frequency.—A rather rare affection, usually occurring between the ages of one and four years, and characterized by its sudden occurrence, febrile character and subsequent hemiplegia and rigidity.

Causes.—None can be assigned. The affection is probably due to the same causes as acute anterior polio-myelitis.

Morbid Anatomy.—The motor centres in the cerebrum and the fibres leading therefrom down into the motor tracts are degenerated so as to cause degeneration of the pyramidal tracts.

Symptoms.—The symptoms in the early stages are like those of poliomyelitis—the onset is sudden, and there is fever and usually convulsions. When these cease hemiplegia is found to be present, and spastic symptoms (rigidity) occur later on. There is an arrest of development, but no reaction of degeneration, and the reflexes are increased.

Diagnosis.—The *diagnosis* is very simple when rigidity develops.

Prognosis.—The *prognosis* as to *life* is usually good; as to complete recovery it is bad.

Treatment.—The *treatment* is purely symptomatic.

GENERAL PARALYSIS OF THE INSANE.

(General Paralysis. General Paresis.)

Definition and Frequency.—A comparatively common disease of the nervous system, characterized by mental weakness and various motor disturbances.

Causes.—1. *Age and sex.* The disease usually occurs in men between twenty and fifty.

2. *Syphilis* is by far the most common cause.

3. *Mental overwork* may be a predisposing cause.

Morbid Anatomy.—1. *Seat.* The anterior part of the brain is chiefly involved, but any part may be affected.

2. *Structural changes.* The nerve cells and fibres undergo degenerative atrophy and there is an increase of connective tissue.

3. *Cord.* Sclerosis of the *posterior* columns is of common occurrence, and sclerosis of the *lateral* columns may occur.

Symptoms.—1. The *onset* is usually gradual.

2. *Mental.* A loss of mental *balance* is usually the first indication; loss of moral sense, “delusions of grandeur” and other forms of mental disturbance may be observed.

3. *Speech.* The *speech* is affected, words being frequently pronounced wrong and used in the wrong connection.

4. *Ocular.* Inequality of the pupils and loss of pupillary reflex are often observed.

5. *Motor.* Tremor of the lips when attempting to speak, ataxia of the limbs, sometimes spastic symptoms and rarely paralysis occur.

6. *Reflexes.* Usually the reflexes are lost (posterior sclerosis), but sometimes they are increased (lateral sclerosis).

Diagnosis.—The *diagnosis* is based on the mental disturbances, combined with the physical symptoms mentioned.

It differs from simple *locomotor ataxia* in the fact that in the latter there are no cerebral symptoms.

Prognosis.—The *prognosis* is unfavorable; the duration may be several years or only a few months.

Treatment.—The treatment consists in complete mental and bodily rest and the use of mercury and iodides.

Confinement in an asylum is advisable in many cases.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

Definition.—*Functional* diseases of the nervous system are those in which up to the present time no essential and characteristic morbid change has been found.

With respect to epilepsy, however, (and perhaps some other diseases of this class) it should be remembered that *epileptiform* attacks may occur from certain well-marked lesions of the cortex of the brain, but these cases are not examples of true epilepsy.

EPILEPSY.

Definition and Varieties.—A disease characterized, as a rule, by convulsive attacks and loss of consciousness. This is the usual form or variety. Other varieties are—

“*Petit mal*,” in which there is no convulsion, but only a sudden and brief loss of consciousness.

Brief attacks of insanity (Samt's Psychological Equivalents).

Causes.—1. *Age.* The disease may occur at any age, but in the great majority of cases it begins before twenty.

2. *Heredity.* A neuropathic tendency, shown by some form or other of nervous disease in the ancestors or near relatives, is present in about one-third of the cases.

3. *Injuries* of the skull, meninges, brain substance and peripheral nerves may occasion epilepsy, but when the brain or its membranes or the skull are involved, it is usually Jacksonian epilepsy and not a simple functional disease.

4. *Emotional disturbances* and possibly excessive mental work may induce attacks in persons who are predisposed to the affection.

5 Probably *syphilis* in some cases.

Symptoms.—A. *Prodromic.* An *aura* is of common but by no means invariable occurrence; it may be (1) motor, (2) sensory, (3) secretory, (4) psychical.

B. *During the attack.* 1. *Mental.* There is loss of consciousness in the ordinary form, and this is followed by coma, and then by sound sleep as a general thing; sometimes by temporary insanity which may last a few moments only or several hours.

2. *Muscular*. Convulsions, at first—for a few seconds—tonic in character and then clonic; the convulsions sometimes begin in one limb and then become general, or they may be general from the beginning.

3. *Sensory*. Sensation is temporarily abolished.

4. *Circulatory*. The circulation is interfered with by the muscular contraction and the pulse is consequently irregular and the face livid.

5. *Respiratory*. The respirations are irregular and stertorous and the patient froths at the mouth. Often there is a *cry* at the commencement, from sudden contraction of some of the muscles.

6. *Excretory*. The urine and fæces are often discharged involuntarily.

C. *Symptoms of "le petit mal"*. In this form there is no convulsion, but a simple and brief loss of consciousness; occasionally there is some sensory disturbance at the same time.

D. *Immediately after the attack* there is usually profound stupor, but there may be temporary insanity and occasionally aphasia.

E. *In the intervals between the attacks* the patient is sometimes perfectly well, but in many cases there is a gradually progressive mental weakness; in children there is arrest of mental development in many cases.

F. *The duration and frequency* of the attacks vary. The usual duration of the convulsion is a few moments; of the stupor an hour or two; but the attacks occasionally come in rapid succession for several hours or days ("status epilepticus"); in other cases the interval between the attacks may be several days or even months or years.

Diagnosis.—1. From *hysteria* by the more complete unconsciousness and the history of the case.

2. From *urinæmia* by the absence of any considerable amount of albumin in the urine and the history of the case.

Prognosis.—The *prognosis* as to life is usually good; but sudden death occasionally occurs during or just after a convulsion.

The *prognosis* as to complete recovery is not usually very good.

Results.—If the disease develops in adult life and the attacks are infrequent there may be no serious mental impairment.

In children there is usually arrest of mental development.

Attacks of insanity may take the place of epileptic seizures or follow ordinary attacks.

Treatment.—1. To *remove the cause*, if possible, by antisyphilitic treatment when necessary, by removing tumors or other irritants of the cortex.

2. To *lessen nervous irritability and spasm*, by the bromides, nitro-glycerine, amyl nitrite, belladonna, borax, antifebrine, sulfo-nal, &c.

3. To *improve the general health* by cod liver oil, zinc, &c.

The *bromides* are by far the best remedies in most cases. The acne which bromine causes may be relieved or prevented in many cases by naphthol or salicylate of bismuth, taken internally, or by arsenic.

Surgical treatment. The removal of tumors, of depressed bone, of cicatrices, &c., is only advisable in Jacksonian epilepsy when the localizing symptoms are well marked.

INFANTILE CONVULSIONS.

Convulsions frequently occur in children from reflex causes (improper food, teething (?), &c.), or in connection with febrile disturbances or the acute infectious diseases; they occur, also, as a rule, in the early stages of acute cerebral paralysis and acute anterior poliomyelitis and in rickets.

They are attended by loss of consciousness and usually a greater or less elevation of temperature.

The *prognosis* depends upon the cause.

The *treatment* is addressed to the removal of the cause, by purgatives, &c., and to lessening nervous irritability, by chloral, the bromides, acetanilide, warm baths, &c.

HYSTERIA.

Definition and Frequency.—A very frequent affection, characterized by the most varied emotional, sensory and motor disturbances.

Causes.—1. *Age and sex.* The disease is most common in young women, but may occur at any age and in either sex.

2. *Neuropathic diathesis.* A tendency to nervous diseases in other members of the family is often found.

3. *Emotional excitement* is a very common exciting cause.

4. *Anemia and debility* are common predisposing causes.

5. *Uterine disease* was formerly thought to be intimately associated with hysteria, but the connection is now considered doubtful.

Symptoms.—1. *Psychical.* *Instability* is the chief characteristic of the psychical symptoms. The emotions are easily excited.

2. *Motor.* *Spasm* of the muscles of the throat (*globus hystericus*) of the limbs and of the trunk is very frequent.

Paralysis is also of frequent occurrence. Both the spasm and paralysis are characterized by their want of permanence.

3. *Sensory.* *Hyperæsthesia* and *anæsthesia* are common symptoms. Ovarian hyperæsthesia is especially frequent.

Precardial pain is present in many cases, and is probably due to spasmodic contraction of the blood vessels.

4. *Reflexes.* The reflexes may be increased or diminished; as a rule, they are increased.

5. *Circulatory.* The pulse is rapid and tense.

6. *Urinary.* At the close of an attack there is usually a flow of limpid urine from the increased blood pressure.

Diagnosis.—1. From *epilepsy*. (See *epilepsy*.)

2. From *puerperal eclampsia* by the absence of albuminuria.

3. From *cerebral tumors* by the absence of choked disk.

4. From *paralysis from organic disease* by the more transitory character of the symptoms.

Prognosis and Duration.—The *prognosis* as to life is always good; relapses are very frequent.

The duration of an attack may be only a few moments, or it may last weeks or months. Most attacks are of short duration.

Treatment.—1. To remove the cause, such as *anæmia*, excitement, &c.

2. To stimulate the will power by moral treatment.

3. To allay nervous irritability by valerian, Hoffmann's anodyne, assafoetida, musk, &c.

4. To improve the general health by arsenic, strychnia, phosphorus, quinine and other tonics.

CATALEPSY.

Definition and Frequency.—A rather rare disease, nearly allied to hysteria, and characterized by loss of consciousness and a peculiar condition of the muscles which causes the limbs to remain for some time in any position in which they may be placed.

Causes.—The causes are the same as those of hysteria.

Symptoms.—1. The onset is sudden and the attacks are *paroxysmal*.

2. *Psychical*. There is loss of consciousness and loss of sensation.

3. *Motor*. The limbs may be placed in the most singular positions and will remain so for some time.

4. The *respirations* are full and slow, and the *pulse* soft and compressible.

Diagnosis.—It is distinguished from *simulated catalepsy* by the length of time that a limb—the arm for instance—will remain extended.

Prognosis.—The *prognosis* is usually unfavorable.

Treatment.—The *treatment* is the same as that of hysteria. An emetic will often stop a paroxysm of catalepsy or hysteria at once.

CHOREA.

Definition, Frequency and Synonym.—A common functional disturbance of the nervous system, characterized by involuntary contractions of certain muscles or groups of muscles, the movements being of considerable extent—differing in this respect from tremor. It is often called St. Vitus's dance.

Causes.—1. *Age and sex*. It is more common in children and young adults than in advanced life; *women or girls* are more often affected than boys.

2. The *neuropathic diathesis* probably has some influence on its development.

3. *Emotional excitement* and *overwork* are probable causes.

4. *Rheumatism* is by far the most prominent cause.

Symptoms.—1. *Psychical*. Occasionally there is some mental weakness, which is temporary in duration.

2. *Motor*. A muscle or a group of muscles will contract and cause jerking movements. Different muscles are affected at different times and at short intervals.

3. *Sensory* disturbances are absent.

Diagnosis.—1. From *epilepsy* by the fact that there is no loss of consciousness in chorea and the spasmodic movements are more limited in extent.

2. From *disseminated sclerosis* by the absence of *intention* tremor and the greater excursion of the movements in chorea.

Duration and Prognosis.—The *duration* is variable; it may last a few weeks or several months. The *prognosis* is nearly always favorable, except in the case of pregnant women.

Results.—*Valvular disease* of the heart is a frequent result.

Treatment.—1. To *allay nervous irritability* by chloral, the bromides, acetanilide, phenacetine, &c.

2. To *improve the general health* by tonics, such as cod liver oil and iron, and especially *arsenic*.

PARALYSIS AGITANS.

Definition and Frequency.—A rather rare affection, confined almost exclusively to persons of advanced age, and characterized by constant tremor.

Causes.—1. *Age and sex.* The disease is rare before sixty and is rather more common in women than in men

2. *Emotional disturbances* increase, if they do not cause the trouble.

Symptoms.—The *symptoms* are exclusively *motor* and consist in *constant tremor* of the hands and head and often of other parts. The *attitude* is striking—the patient bending forward and showing a tendency to rush forward on the slightest push from behind.

Diagnosis.—From *disseminated sclerosis* by the absence of intention tremor.

Duration and Prognosis.—When once developed, the disease persists through life, but the prognosis is favorable, so far as life itself is concerned.

Treatment is useless.

WRITERS' CRAMP.

Definition and Frequency.—A rather rare affection, characterized by cramp of the muscles used in writing when such work is attempted.

Cause.—The only cause is *overwork* of the muscles involved. Possibly the *neuropathic diathesis* has some influence on its production.

Symptoms.—1. *Sensory*. There is a feeling of weight and stiffness in the affected muscles.

2. *Motor*. The muscles used in writing undergo spasmodic contraction whenever such work is attempted.

Diagnosis.—1. From *lead palsy* by the absence of any paralysis of the extensors of the hand and of other evidences of lead poisoning.

2. From *insular sclerosis* by the absence of tremor and the more marked spasm of the muscles.

Duration and Prognosis.—The *prognosis* as to life is of course good, but is doubtful as to recovery, and the affection *may* last through life.

Treatment.—1.— *Absolute rest* to the muscles involved is imperative. Mechanical appliances to hold the fingers apart in writing are useful.

2. *Galvanism* and *massage* are sometimes beneficial.

SUNSTROKE.

(Insolation.)

Causes.—1. *Heat*, especially moist heat, which prevents evaporation from the skin.

2. *Auxiliary causes* are exhaustion, stimulants and over-eating.

Symptoms.—A. *Prodromic* symptoms, which do not always occur, are headache and dizziness.

B. The symptoms of a *developed attack* may occur in three forms, which are usually combined—

1. *Heat prostration*, in which the skin is cool, the pulse feeble and consciousness is retained.

2. *Sunstroke proper*, in which the symptoms are much like those of heat prostration, except that there is loss of consciousness and shallow respiration.

3. *Thermic fever*, which is characterized by very high temperature, often 109° or 110° , a full and slow pulse, later becoming rapid and feeble, and profound coma.

Diagnosis.—The *diagnosis* is based on the history and usually presents no difficulty.

Prognosis and Results.—About one-half of the persons affected with sunstroke die.

Heat prostration is the least and *thermic fever* the most dangerous. Recovery from any form is slow, the patients being liable to severe headache for months afterwards, and occasionally insanity results.

Treatment.—1. In *heat prostration* and *sunstroke* proper the most important point is to sustain strength by ammonia and brandy, the latter being administered with great caution.

2. In *thermic fever*, the reduction of temperature is of the first importance. The cold bath, applications of ice, and antipyrine hypodermically, are the best agents for this purpose.

NEURASTHENIA.

Definition.—A functional affection characterized by defective strength and tone of the nervous system.

Causes.—1. *Age and sex.* The affection is most common in men in young adult or middle life.

2. *Sexual excesses, excessive study, emotional excitement,* and the excessive use of alcohol and tobacco are probable causes.

3. The *neuropathic diathesis* predisposes to neurasthenia.

Symptoms.—1. *Cerebral.* A feeling of pressure about the head and inability to do prolonged mental work are the chief cerebral symptoms.

2. *Motor.* There is usually muscular weakness, and the patient is easily fatigued.

3. *Sensory symptoms,* other than headache and sometimes pain in the eyes, are not usually very marked.

4. *Genital.* Diminution or loss of sexual power is a common symptom.

Diagnosis.—1. From *organic disease* of the cord by the absence of the symptoms of such disease.

2. From *cerebral tumor* by the absence of choked disk.

Prognosis and Duration.—The *prognosis* as to life is good,

The *duration* very uncertain, and relapses are frequent.
It does not lead to organic disease.

Treatment.—1. To *remove the cause* by mental rest, the avoidance of excitement and the removal of injurious influences.

2. To *improve nutrition* of the nervous system, by life in the open air, the administration of tonics, such as iron, strychnia, cod liver oil, phosphorus and arsenic and the use of galvanism and massage.

SICK HEADACHE.

(Megrim. Migraine.)

Causes.—1. *Age and sex.* Sick headache is rather more common in women than in men, and occurs usually in youth and early adult life.

2. *Heredity*, or the occurrence of some form of nervous trouble in previous generations, can usually be determined.

3. *Emotional excitement and digestive disturbances* are frequent causes.

4. *Anomalies of refraction* frequently cause attacks of severe headache.

Symptoms.—1. *Special senses.* Disturbances of vision are very common at the beginning of an attack.

2. *Sensory.* The headache is usually intense and there is often a feeling of stiffness in the muscles.

3. *Digestive.* Nausea is a common, but by no means an invariable symptom. Vomiting may occur often just at the close of an attack.

4. *Vaso-motor.* As a rule, the face is pale, but in some cases there is flushing.

5. *Urinary.* Frequently at the close of an attack there is a profuse flow of limpid urine from the increased blood pressure during the attack.

Diagnosis.—The *diagnosis* is easy and needs no comment.

Frequency, Duration and Prognosis. The attack may occur every few days or at intervals of weeks or months. The *duration* is usually several hours and often the termination is sudden.

The prognosis as to life is good and the attacks usually become less frequent with advancing years.

Treatment.—1. To *remove the cause*, when possible, by correcting errors of diet, avoiding excitement or overwork and the use of proper glasses when necessary.

2. To *correct circulatory disturbances*. In some cases where there was flushing of the face ergot has been found useful, and when pallor exists amyl-nitrite and nitro-glycerine are beneficial.

3. To *lessen the pain*, phenacetine, acetanilide, antipyrine, guarana, potassium bromide, codeia, &c., have been used. Few cases fail to yield to phenacetine, antipyrine or guarana.

SEA-SICKNESS.

Sea-sickness may be caused by any unusual motion, and is characterized by great prostration and intense nausea.

The *prognosis* is nearly always favorable, but in long voyages death may occur from exhaustion.

The *treatment* consists in the administration of champagne and other stimulants, hydrocyanic acid, the bromides, amyl nitrite and nitro-glycerine.

EXOPHTHALMIC GOITRE.

(Graves's Disease. Basedow's Disease.)

Definition.—A disease characterized, when fully developed, by swelling of the thyroid gland, protrusion of the eyeballs and rapid action of the heart.

Causes.—1. *Age and sex.* The affection is far more common in women than in men, and occurs usually between the ages of twenty and forty.

2. The *neuropathic diathesis* is a predisposing cause.

3. *Emotional excitement and menstrual disturbances* increase, if they do not cause the affection.

Symptoms.—1. *Cardiac.* The pulse is very rapid, often 130 to the minute, and is usually full and strong. Irregularity is not common.

2. *Thyroid.* The thyroid gland is enlarged, but not usually to a very great extent.

3. *Ocular*. The eyes protrude and when the patient looks down, in some cases, the upper lid does not follow the ball (von Græfe's symptom.)

Diagnosis.—The *diagnosis* is based on the simultaneous occurrence of rapid action of the heart, thyroid enlargement and protrusion of the eyes.

Prognosis and Duration.—The *prognosis* as to life is good. The *duration* is long and complete recovery rare.

Treatment.—The *treatment* consists in the administration of digitalis and ergot and the use of galvanism applied to the goitrous enlargement and the sympathetic nerves in the neck.

TETANY.

(Intermittent tetanus.)

Definition and Frequency.—A very rare affection of the nervous system, characterized by intermittent spasms of certain groups of muscles.

Causes.—The disease is more common in children and young adults and, especially, in nursing women. Cold is sometimes a cause.

Symptoms.—The *prodromic* symptoms are slight pain and stiffness in the muscles.

During the attack there is a tonic contraction of some of the muscles of both upper extremities, as a rule. There are *no sensory disturbances*, but the irritability of the muscles is greatly increased, and a slight blow upon them will cause contraction.

The attacks vary greatly in frequency and duration. They are usually several days or weeks apart, but the paroxysms may occur every few moments. The attack (not the paroxysm) may last for several weeks.

Prognosis.—The *prognosis* is usually favorable.

Treatment.—The *treatment* consists in the use of an ascending galvanic current and the administration of bromides or other nerve sedatives.

ATHETOSIS.

Athetosis is a rare and peculiar affection of the nervous system, characterized by constant movement of certain muscles; those which move the fingers are most commonly involved, but the muscles of the face and neck and also of the trunk and lower limbs may also be involved.

The *causes* are unknown. The disease often occurs after hemiplegia.

The *symptoms* have already been mentioned under the definition. The movements are constant, except during sleep.

The *prognosis* as to life is good; as to recovery bad.

No *treatment* has been found of any avail.

APPENDIX.

[The diseases treated of in this appendix were accidentally omitted from their proper position.]

MEASLES.

(Rubeola.)

Definition and Frequency.—An acute, infectious and very contagious disease, characterized by coryza and the occurrence of a papular eruption. It is of great frequency.

Varieties.—1. *Simple* measles, in which the case runs a mild course.

2. *Malignant* or *black* measles, in which the eruption is very dark in color, from great congestion and the case runs a severe course.

Causes.—1. *A germ* which has not been isolated.

2. *Media of contagion*, the atmosphere, clothing, letters, &c., the germs being contained in the mucous secretions, blood and epithelial scales.

3. *Avenues of introduction*, the respiratory passages and, possibly, the skin (by inoculation). It is infectious at all stages.

4. *Immunity* is conferred by one attack, as a rule.

5. *Age*. It may occur at any age, but children are more liable to it, probably because they are not protected by a previous attack.

6. The *period of incubation* is about ten days.

Morbid Anatomy.—1. Changes in the *larynx* and *bronchi*. The mucous membrane of the larynx and bronchi and also that of the nose is inflamed.

2. The *conjunctivæ* are also inflamed.

3. The *changes in the skin* will be described under symptoms.

Symptoms.—A. *Prodromic*. 1. *Coryza* is a very prominent prodromic symptom; *bronchitis* is also present and persists after the eruption comes out.

2. *Temperature*. The temperature is elevated, usually about 102° to 104°.

B. *Eruptive stage*. 1. *Characteristics of the eruption*; it appears on the *fourth day*, is *papular* in character, appears first on the *face*, is usually bright red in color and each *papule* is about one-eighth of

an inch in diameter; the rash reaches its height on the third day and disappears by the sixth.

2. The *temperature* is about 104° or 105° and falls quite suddenly when the rash has been out about two days.

3. *Respiratory*. Cough persists during the eruptive stage.

4. *Digestive*. Nausea and vomiting are common symptoms and diarrhœa may occur as a complication.

C. *Desquamative stage*. *Desquamation* occurs in the form of small scales and a severe cough (the "iron cough") lasts for some time after the eruption has disappeared.

Irregular Attacks.—Attacks of measles may occur without any or with but very slight eruption.

Black measles is a severe type, in which the eruption is very extensive and the congestion of the skin great.

Diagnosis.—1. From *scarlet fever* by the premonitory coryza and the absence of sore throat and by the papular character of the rash.

2. From *small pox* by the coryza and the character of the eruption.

Prognosis.—The *prognosis* is good in uncomplicated cases. It is dependent on (1) *hygienic surroundings*, over-crowding adding to the danger; (2) *temperature*, the higher the temperature the greater the danger; (3) the *character of the rash*; (4) *age*, it is more serious in *adults* than in children; (5) *previous health*, and (6) *complications* (q. v.)

Complications.—1. *Capillary bronchitis* from extension of inflammation to the smaller tubes.

2. *Catarrhal pneumonia* from extension, the inhalation of noxious products from the tubes and probably also from the presence of leucomaines.

3. *Inflammation of serous membranes* and *nephritis* are rare in measles, but sometimes occur from the action of leucomaines.

4. *Affections of the eyes and ears*. *Conjunctivitis* is common, and is often quite severe; *otitis media* is also common, and frequently a severe complication; it results from the passage of germs up through the Eustachian tube.

5. *Intestinal catarrh* from the elimination of leucomaines by the mucous membrane of the intestines.

6. *Acute tuberculosis* probably because the lungs are weakened and less able to resist the action of germs.

Treatment.—A. *Prophylactic*. The only prophylactic is *isolation*.

B. *Medicinal*. 1. To *avoid* chilling and prevent complications by confinement to bed and the avoidance of draughts.

2. To *prevent injury to the eyes*, by keeping the room dark and prohibiting reading.

3. To *reduce hyperpyrexia* and *relieve headache*, by phenacetine and other drugs of its class.

4. To *relieve bronchitis*, by vapor inhalations and the administration of ipecac, squills or similar expectorants, with opiates in small doses.

GERMAN MEASLES.

(Rotheln. Rubella.)

Definition.—An acute infectious and moderately contagious disease, characterized by a rash, papular in character, but the papules are smaller than those of measles.

Causes.—1. A *germ* probably, but it has not been isolated.

2. The *medium of contagion* is probably the atmosphere, but the disease is apparently not very contagious.

3. The *avenue of introduction* is probably the respiratory mucous membrane.

4. *Children* are more liable to the disease than adults.

5. Immunity is conferred by one attack.

6. The *period of incubation* is from ten days to three weeks.

Morbid Anatomy.—There is no characteristic morbid anatomy, but the glands at the back of the neck are nearly always enlarged.

Symptoms.—The *subjective* symptoms are usually very slight.

1. The *eruption* is often the first symptom; it is in small papules, red in color, and lasts about two days; desquamation seldom, if ever, occurs.

2. The *temperature* is very little elevated.

3. The *eyes* are frequently inflamed, and are left weak when the attack is over.

4. The *glands* at the back of the neck are enlarged.

Diagnosis.—1. From *measles* by the absence of coryza and the smaller size of the papules.

2. From *scarlet fever* by the absence of sore throat and the papular character of the eruption.

Prognosis.—The *prognosis* is uniformly good.

Treatment.—The *treatment* is purely symptomatic. Entire

rest to the eyes is important. If headache is severe, as occasionally happens, phenacetine may be used with advantage.

SMALL-POX.

(Variola.)

Definition.—An acute infectious and very contagious disease, characterized by the occurrence on the skin and mucous membranes of an eruption, first macular, then papular, vesicular and pustular, the pustules being umbilicated.

Varieties.—1. *Discrete* when the pustules are scattered.

2. *Confluent* when the pustules run together, forming a more or less continuous mass of pustules or scabs.

3. *Hemorrhagic* in which the exudate is blood instead of pus; this is the most severe form.

Causes.—1.—*A germ*, which has not been isolated.

2. The *favorable condition for development* is overcrowding, but the disease is highly contagious under all circumstances and at all stages.

3. The *avenues of introduction* are (1) an *abraded surface*; (2) the *respiratory mucous membrane*.

4. The *media of contagion* are the air, clothing, persons sick with the disease, &c

5. *Immunity* is nearly always conferred by one attack.

6. *Age*. It may occur at any age, even in intra-uterine life.

7. The *period of incubation* is from five to thirty days unless the disease is inoculated, when it is about forty-eight hours.

Morbid Anatomy.—1. The *internal organs* are congested, the spleen being enlarged considerably, probably from the action of the leucomaines and also from the inability of the skin to do its proper work.

2. The *skin* shows an eruption at first macular and then successively papular, vesicular and pustular. The whole thickness of the skin is involved, as a rule; the individual spots are from $\frac{1}{8}$ th to $\frac{1}{4}$ th of an inch in size, and the skin at the point infiltrated with serum and cells. In the vesicular stage the eruption becomes umbilicated from the exudate around the circumference of the spots, the centre being held down by flattened epithelial cells. Each pustule

is divided into several compartments by the framework of epithelial cells.

3. The *mucous membrane* undergoes changes similar to those in the skin.

Symptoms.—1. *Nervous.* A chill, headache, very severe backache, delirium, restlessness and somnolence. All these symptoms are probably due to the poisoning by leucomaines and to the fever.

2. *Temperature.* The temperature is elevated at first— 102° to 104° —but usually falls on the fourth day, when the eruption appears. On the eighth or ninth day, when suppuration occurs in the pustules, there is another rise of temperature, often to 105° or even 109° , which is due to the absorption of septic matter. It declines gradually in favorable cases and usually disappears by the fourteenth or fifteenth day.

3. The *circulatory* symptoms are rapid pulse, which is due to the fever, and later, weakness of the heart's action, from albuminoid degeneration and the prostration caused by weakness.

4. The *digestive* symptoms are nausea and vomiting and sore throat—the former due probably to the action of leucomaines and the swallowing of morbid matters, and the latter (sore throat) to the eruption in the throat.

5. *Cutaneous.* Sweating usually occurs in the early stages.

The *eruption* begins on the *face* usually. It appears on the fourth day, and has the character already mentioned under morbid anatomy. The pustules dry and form crusts, which come off from the fourteenth to the eighteenth day and usually leave pits.

Usually a macular eruption appears before the real rash of small pox, but it lasts only a short time.

Diagnosis.—1. From *measles* by the violent backache, the absence of marked coryza, and above all by the rash which soon becomes vesicular and then pustular.

2. From *typhus fever* by the character of the eruption.

In many cases it is impossible to make a diagnosis in the early stages before the vesicles and pustules appear.

Complications.—1. *Pulmonary and laryngeal.* Broncho-pneumonia from the inhalation of morbid matter and ulceration and possibly stenosis of the larynx from the ulceration.

2. *Special sense.* Otitis media may occur from extension from the throat. Occasionally keratitis and ulceration of the eyes occurs.

3. *Urinary.* Albuminuria is common, and occasionally well-marked nephritis occurs.

Prognosis.—The *prognosis* is always serious, and is influenced by the following circumstances:

1. *Amount of eruption* and *type* of the disease; *varioid* is least dangerous; *discrete* small-pox is less dangerous than *confluent*, and hemorrhagic is most dangerous.

2. *Intemperate habits* increase the danger.

3. *Previous good health* renders the prognosis more favorable.
 4. *Pregnancy* adds greatly to the danger.
- The time of greatest danger is the eighth day.

Causes of Death.—1. *Toxæmia*, from the amount of leucomaines absorbed.

2. *Exhaustion*, from extensive suppuration and poisoning.

Treatment.—A. *Prophylactic*. 1. *Vaccination* (q. v.)

2. *Quarantine*. To be protective the quarantine must be thorough and all fomites must be destroyed or thoroughly disinfected.

B. *Medicinal*. 1. To *reduce temperature* by phenacetine, &c.

2. To *allay restlessness* by the bromides, phenacetine, codeia and other analgesics.

3. To *promote eruption* when slow in appearing, by warm baths and Dover's powder.

4. To *sustain strength* by nutritious food, and stimulants if exhaustion is imminent.

5. To *prevent pitting* by the application of a paste of carbolic acid, glycerine and prepared chalk.

(There is no satisfactory method of preventing pitting.)

During desiccation *warm baths* with subsequent oiling of the surface should be employed to promote the removal of the crusts.

Varioloid is a mild form of small-pox which occurs usually in those who are partially protected by vaccination. In itself it is not dangerous, but it may cause severe small-pox in others who are unprotected.

VACCINATION.

Varieties of Virus.—1. *Non-humanized*, which is obtained directly from the cow pox pustules on the udder, and

2. *Humanized*, which is obtained from a vaccination pustule or scab on the human being.

Protective Power.—Persons properly vaccinated are almost entirely protected from the danger of small pox, but the immunity does not last indefinitely, so that vaccination should be repeated every three or four years.

Relative Advantages of Bovine and Humanized Virus.—

Bovine virus causes a more severe sore, as a rule, than humanized, but it is more satisfactory in its results, and there is no danger of communicating other diseases when it is employed.

Method of Procedure.—The skin should be slightly scratched or pricked, so as to get through the outer layers of the skin, but bleeding should be avoided because the blood may wash out the virus. Having scratched the skin, the virus is to be rubbed over the surface. If a "vaccine point" is used, the end of the point on which the virus is must be moistened with water first and then rubbed on the scratched or punctured surface.

Changes at and Around the Vaccinated Spot.—For several days there is no inflammatory reaction; on the fourth or fifth day it commences to inflame, and a vesicle, and subsequently a pustule, is formed by the eighth or ninth day; the pustule is umbilicated, and dries into a crust which comes off on the fourteenth day.

Circumstances which interfere with the success of vaccination are (1) erysipelas and (2) certain acute and chronic skin diseases. Some people are unsusceptible to it.

Diseases Which may be Communicated by Vaccination.—

Syphilis and possibly *tuberculosis* may be communicated by vaccination with humanized virus, but such accidents are *extremely rare*.

CHICKEN-POX.**(Varicella.)**

Chicken-pox is an eruptive disease which occurs usually in children. The eruption is in the form of pustules which are usually very few in number, and which lead to pitting.

It is contagious and is much more common in children than in adults.

The *symptoms* are seldom marked. There may be slight fever and malaise, and sometimes there is much itching of the surface around the pustules, which resemble those of small-pox, being usually umbilicated.

The *prognosis* is always favorable, and the *treatment* symptomatic.

If the itching is severe, dusting with starch will usually give relief.

CEREBRO-SPINAL MENINGITIS.

Cerebro-Spinal Fever.

Definition and Frequency.—A rather rare disease, prevailing as an epidemic and characterized anatomically by inflammation of the cerebral and spinal meninges.

Causes.—1. A *germ* is almost certainly the essential cause, but it has not been isolated with absolute certainty. It is probable that it is a diplococcus similar to, if not identical with, that of lobar pneumonia (q. v.)

2. Nothing definite is known with respect to the *favorable conditions of development* or the avenues of introduction.

3. *Age.* The disease may occur at any age, but is more common between ten and twenty.

4. It is *very slightly contagious* and defective hygienic conditions have little to do with its development or extension.

Morbid Anatomy.—1. The *meninges of the brain* are inflamed and there is a purulent exudate which is especially marked around the vessels. The brain itself may be infiltrated and softened at certain spots.

2. The *meninges of the cord* are also inflamed; the roots of the spinal nerves are frequently affected in a similar way.

3. The *liver, spleen, heart and kidneys* show albuminoid degeneration, from the action of the leucomaines.

Symptoms.—1. The *onset* usually is sudden, but may be gradual and marked by stiffness in the back and shoulders and a feeling of malaise.

2. *Nervous.* The chief nervous symptoms in the *early stages* are headache, backache, stiffness of the back and possibly opisthotonos, from the increased irritability of the sensory nerves and reflexes, and vertigo and delirium, which are probably due to poisoning by ptomaines. *Convulsions* are quite common, especially in young patients.

At a *later stage* coma and paralysis appear, from the leucomaine poisoning and pressure of the exudate, or else from inflammation and destruction of nerve tissue.

3. *Special senses.* *Photophobia*, from increased sensibility of the sensory nerves; unequal pupils, from increased irritability or pressure of the exudate on some of the nerves; ptosis and strabismus, from the same cause, are common symptoms. Later on there may be pan-ophthalmitis. *Deafness*, from involvement of the acoustic nerve or from otitis media occurs in some cases. *Loss of the sense of taste and smell* is occasionally observed.

4. The *temperature* is usually but little elevated, but may be high.

5. The *pulse* is variable as to frequency and force, the variability being due probably to irregular nervous supply.

6. The *skin* is hyperaesthetic from the increased sensibility of the sensory nerve roots and the nerve centres themselves, and there is an eruption of herpes on the lips and face in most cases. Sometimes there is a macular or petechial rash also.

7. *Nausea, vomiting* and *anorexia* are nearly always observed and are probably central in origin.

8. *Albuminuria* is occasionally present, but well-marked nephritis is not common.

Diagnosis.—1. From *small pox* by the absence of the characteristic eruption of the latter disease.

2. From *tubercular meningitis* by the history of the case and the more marked spinal symptoms in epidemic meningitis.

3. From *acute myelitis* by the absence of the symptoms of irritation (spasms and hyperæsthesia) in the latter.

Prognosis.—The *death rate* is from 30 to 80 per cent. The prognosis is dependent on the *age* of the patient, the *character and period of the epidemic* and on the occurrence of *complications*.

The *duration* is variable; it is usually about two weeks, but the disease may last for months.

Complications.—1. *Pulmonary.* *Lobar pneumonia* is quite common in connection with this disease. *Lobular pneumonia* also occurs.

2. *Cardiac.* Attacks of sudden and alarming heart failure occur, probably from defective supply of nerve force.

Causes of Death.—1. *Toxæmia*, from the absorption of leucomaines.

2. *Coma*, from pressure of the exudate or poisoning by leucomaines.

3. *Paralysis* or *spasm* of respiratory muscles.

4. *Asthenia*, from defective supply of nerve force or from exhaustion.

5. *Complications* (q. v.).

Sequelæ.—1. *Mental* and *nervous*. Weakness of intellect and paralysis or paresis of certain muscles frequently result.

2. *Special senses.* *Blindness*, from pan-ophthalmitis, and *deafness*, from involvement of the acoustic nerve or from otitis media, are not uncommon sequelæ.

Treatment.—A. *Hygienic.* The hygienic treatment is the same as that of typhoid fever.

B. *Medicinal.* 1. To *relieve pain* by quiet, opiates, phenacetine, bromides and cold or hot applications to the spine.

2. To *relieve symptoms*, such as thirst and constipation, by appropriate remedies.

3. To *promote absorption*, after the acute stage has passed, by iodide of potassium and blisters.
 4. To *sustain strength* by nourishment and stimulants.
-

WHOOPIING COUGH.

(Pertussis.)

Definition and Frequency.—An acute, infectious disease, of great frequency and characterized by attacks of violent coughing, spasmodic in character and attended by a peculiar whoop.

Causes.—1. A *germ* is almost certainly the essential cause, but it has not yet been found.

2. The *favorable conditions* for development are (1) *age* of the patient—children under ten being far more liable to it than older persons; and (2) *previous health*—it is especially common after epidemics of measles and in teething children.

3. The *media of contagion* are the air, and probably clothing; the infectious agent is in the discharge from the air passages.

4. The *avenue of introduction* is the respiratory tract.

5. *Immunity* is conferred by one attack in nearly all cases.

6. The *period of incubation* is from five to fourteen days

Morbid Anatomy.—The *morbid anatomy* is not characteristic. The bronchi are inflamed and the bronchial glands are usually enlarged.

Symptoms.—A. In the 1st, or *catarrhal* stage, which lasts from one to three weeks, the symptoms resemble those of ordinary bronchitis.

B. In the 2d, or *spasmodic stage*, there are attacks of violent coughing, in which the patient “loses his breath,” and recovers it with a long-drawn inspiration or whoop.

Vomiting frequently occurs during a paroxysm of coughing, and the face becomes livid.

Occasionally there are *extravasations of blood* into the conjunctiva, which, however, are not serious.

Urine and feces may be passed involuntarily during a “fit” of coughing.

This stage usually lasts about *three weeks*, and is followed by the

C. Third stage, or stage of decline, in which the symptoms gradually abate. This stage lasts from three to six weeks.

Diagnosis.—The *diagnosis* is based on the prevalence of an epidemic and the character of the cough.

Prognosis.—The *prognosis* is usually favorable unless complications arise.

Complications.—1. *Pulmonary.* *Collapse of the lung and catarrhal pneumonia* are very common. *Emphysema* is an occasional sequel and *tuberculosis* is quite a common sequel.

2. *Nervous.* *Cerebral hemorrhage*, with consequent paralysis, is a very rare complication.

Treatment.—1. To *lessen the violence of the paroxysms* by belladonna, bromides, chloral, chloroform, musk, cocaine, carbolic acid by inhalation, antipyrine, phenacetine and quinine.

2. To *improve the general health* by arsenic and quinine and other tonics.

3. To *prevent complications* by the avoidance of cold and draughts, and especially of over-crowding. Fresh air is very important.

‘HYDROPHOBIA.

(Rabies, when it occurs in animals.)

Definition and Frequency.—An acute infectious disease, of great rarity in this country, characterized by peculiar spasms of the muscles of deglutition and sometimes of respiration.

Causes.—1. A *virus*, probably a *germ*, but the nature of the poison has not been definitely determined.

2. The *avenue of introduction* is always through the broken skin and the *medium of conveyance* is the *saliva* of a *rabid animal*.

3. The *period of incubation* is usually from three to six months, but it varies within still wider limits.

Morbid Anatomy.—There is no characteristic morbid anatomy. Occasionally the nerve centers and the respiratory organs are found congested.

Symptoms.—A. *Incubative.* Feverishness, loss of appetite and depression are the usual symptoms of the prodromic period.

B. In the *second or convulsive* stage there are, at intervals of about half an hour, *spasmodic contractions* of the muscles of *deglutition* and *respiration*. The spasm of the muscles of deglutition is greatly increased by attempts to swallow; there is great thirst.

The mind often remains clear, but occasionally delirium supervenes before death.

Diagnosis.—1. From *tetanus* by the absence of trismus and opisthotonos and the more limited character of the convulsions.

2. From *hysteria* by the greater gravity of the symptoms.

Prognosis.—The *prognosis* is nearly always bad.

Causes of Death.—1. *Asphyxia*, from spasm of the respiratory muscles.

2. *Exhaustion*, from inability to take food.

Treatment.—A. *Preventive Inoculation*, according to the method of Pasteur, with attenuated virus. The virus is obtained from the nervous matter, spinal cord usually, of a rabid animal. The virulence is diminished by keeping it, and the person who has been bitten is inoculated first with the most attenuated and subsequently with virus of gradually increasing virulence.

B. *Remedial*. Temporary *ligation* of the limb above the bitten point and *sucking of the wound* to withdraw the virus is advisable. *Curare* has been used in a very few cases with success.

Chloroform, chloral and other anti-spasmodics have been employed without benefit.

DENGUE.

(Dandy Fever. Breakbone Fever.)

Definition.—An infectious disease, characterized by severe pain, fever and some swelling of the joints, and prevailing as an epidemic.

Causes.—1. A *germ* is in all probability the essential cause.

2. The *favorable conditions* for development are *warmth* and *slight elevation*. It is but little, if at all, contagious and it is doubtful whether one attack confers immunity.

3. The *period* of incubation is from three to five days.

Symptoms.—The *general appearance* is striking, from the stiffness of the muscles.

2. The *nervous symptoms* consist in violent pain, chiefly in the joints, but often in other parts of the body. The *onset* of these pains is usually sudden.

3. The *temperature* is elevated, often to 104° or 105° , and the *pulse* is quick.

5. *Anorexia* is the rule, and *nausea* and *vomiting* occasionally occur.

5. The *joints* are swollen, tender and painful, and the *glands* are often enlarged.

6. In many epidemics there is a *rash* resembling that of scarlet fever, but in many cases it is absent.

7. A *remission* occurs usually in from two to four days, which is followed by another paroxysm.

Duration and Prognosis.—The usual *duration* of an attack is about eight days, but recurrences are frequent.

The *prognosis* is almost uniformly favorable.

Diagnosis.—1. From *remittent fever* by the greater intensity of the joint pain and the eruption.

2. From *rheumatism* by the epidemic character and the involvement of the smaller joints.

Treatment.—1. To *relieve pain* by phenacetine, codeia, salicylate of soda, &c.

2. To *relieve symptoms*, such as constipation and nausea.

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